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GASTRITIS: A STUDY OF 112 CASES DIAGNOSED BY GASTRIC BIOPSY.[1]

By R. K. DOIG and I. J. WOOD,

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NUMEROUS divergent opinions are current on the relation between gastritis and dyspeptic symptoms. At one extreme, Ricketts, Palmer and Kirsner (1949), while recognizing various types of gastritis, find no relation between symptoms and gastroscopic findings. At the other extreme, Schlindler (1947) details separate clinical pictures for each variety of gastritis. Much of this difference of opinion can be related to the varied interpretations of appearances seen through the gastroscope, and many consider that more caution must be observed (Symposium on Symptomatology of Chronic Gastritis, 1943). That such caution is needed can be confirmed by comparing the varying reports of gastroscopists in examining patients with pernicious anæmia, and the almost uniform report by histologists of atrophy of the specific gastric glands (Molofsky and Hollander, 1951).

With the development of the technique of blind gastric biopsy (Wood *et alii*, 1949*a*; Wood, 1951) we have felt that the problem should be reviewed by a study of cases in which the diagnosis of gastritis has been made on histological grounds. The purpose of this paper is to report the clinical findings in a group of 112 cases of gastritis so diagnosed.

[1] Aided by a grant from the National Health and Medical Research Council of Australia.

Method of Study.

The patients have been studied by routine history taking and physical examination, and by gastric biopsy and fractional test meal examination. The cases include all those diagnosed between April, 1948, and December, 1950, and have been selected on the basis of the presence of histological evidence of gastritis and the absence (in the majority) of radiological evidence of gastric or duodenal ulcer or gastric carcinoma. Cases in which there were also gall-stones or other extragastric lesions have been included.

The cases have been arbitrarily subdivided into two groups—superficial and atrophic gastritis—according to whether the amount of atrophy of the gastric glands appears to be less than or greater than half the normal complement of specific cells (Motteram, 1951). There were 48 cases of superficial gastritis and 64 cases of atrophic gastritis. In material from 769 biopsies on 504 patients we have not encountered the condition of hypertrophic gastritis. Cases of gastric atrophy (Doig and Wood, 1950; Motteram, 1951), as seen in association with pernicious anæmia, have been excluded, although five cases of pernicious anæmia or subacute combined degeneration of the cord with atrophic gastritis have been included. In considering gastritis we have paid attention only to the acid and pepsin secreting area. Post-mortem studies by Hebbel (1949), Magnus (1946), Faber (1935) and Wynn Williams (1950) all show that when this area is involved the lesion is diffuse, except when cancer or ulcer is present; and a review of our material agrees with this (Wood *et alii*, 1949*b*; Funder and Weiden, 1952). We have not included in this survey lesions of the antral region.

The cases have been investigated in the Clinical Research Unit and the majority followed for a variable period, ranging up to three years. Comparable information on a

similar group of cases of peptic ulcer, investigated and followed in the unit, has been analysed in a similar fashion (Doig and Wood, 1952).

Age and Sex Distribution.

The age of the patients with gastritis at the development of symptoms and at the time of reporting to hospital is compared with the age of patients with duodenal and gastric ulcer in the histograms of Figure I. Patients with duodenal ulcer develop symptoms at a much earlier age and are seen at hospital earlier than those with gastric ulcer and with gastritis. The average ages of onset in gastric ulcer and gastritis are not significantly different one from another; but when examined at hospital patients with atrophic gastritis are older than those with superficial gastritis. The average age of patients with gastric ulcer lies between the two and the differences are not

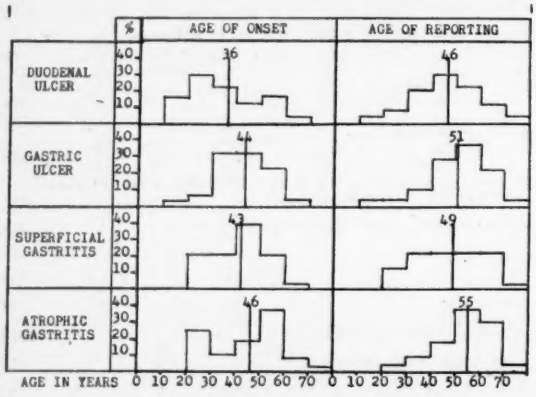

FIGURE I.

Age distribution (histogram) and mean value (vertical line) in cases of duodenal ulcer, gastric ulcer, superficial gastritis and atrophic gastritis showing the age of onset and age of reporting to hospital. The ages for duodenal ulcer are significantly less than the ages for the other three groups. The age of onset of superficial gastritis and atrophic gastritis are the same, but when examined at hospital patients with atrophic gastritis are older than those with superficial gastritis; this suggests that atrophic gastritis is an advanced stage of superficial gastritis.

significant. (Differences are taken as significant when probability is less than 0·05 by the *t* test.) These findings suggest that gastric ulcer is more closely related to gastritis than to duodenal ulcer, and that atrophic gastritis is a more advanced stage of superficial gastritis.

Of the patients with superficial gastritis 12 were males and 36 females; of those with atrophic gastritis 28 were males and 36 females. Chronic alcoholism partly accounted for the relatively greater number of males in the atrophic gastritis group (atrophic gastritis: males six, females one; superficial gastritis: males four, females eight).

Ætiology.

No single ætiological factor was apparent. Of the causes suggested in text-books we have not been able to demonstrate a relation to mechanical, thermal or chemical irritation by food, or to oral or nasal sepsis. Nor was it possible to attribute the onset of symptoms to an acute illness of which gastritis might have been a part. Chronic renal failure, congestive cardiac failure, pulmonary tuberculosis, diabetes, and occupational contact with chemical substances have been found infrequently or not at all. Syphilis was present in five cases, yet the symptoms of dyspepsia, the histological appearance of the stomach and the response to specific therapy did not suggest a syphilitic lesion in the stomach. The irregular incidence of hypertension resulted mainly from the fewer patients in the group with gastric ulcer despite their higher average age. It bore no apparent causal relation to the gastritis. In four cases the initial diagnosis was anæmia of obscure type.

In four cases of superficial gastritis and ten of atrophic gastritis there was a history of gall-stones. In four more of the latter group there had been a previous cholecystectomy. Because of these small numbers the significance of the presence or absence of gall-stones cannot be assessed.

The most important factor to be considered was chronic alcoholism (see Table I). Of 48 patients with superficial gastritis 12 were judged to be chronic alcoholics (four males, eight females), and of 64 with atrophic gastritis seven were judged to be chronic alcoholics (six males and one female). Gastric biopsy in patients with chronic alcoholism suggests that chronic alcoholism is associated with gastritis through the related malnutrition rather than through the direct effect of alcohol on the mucosa. This will be discussed elsewhere (Joske *et alii*, 1952). Apart from chronic alcoholism we found little evidence of chronic malnutrition as a cause of gastritis.

However, lack of appetite caused many patients to take a less than optimal diet, and this made it essential to prescribe a full diet as part of the treatment.

TABLE I.

The Ætiological and Associated Features in Gastritis and Ulcer.[1]

Ætiological and Associated Features.	Duodenal Ulcer.	Gastric Ulcer.	Superficial Gastritis.	Atrophic Gastritis.
Total number of cases ..	93	48	48	64
Estimated age of onset in years (average)	36	44	43	46
Age in years at first hospital visit	46	51	49	55
Sex ratio (males : females) ..	4 : 1	2 : 1	1 : 3	2 : 3
Psychological factors — alcoholic patients excluded	58	18	25	21
Chronic alcoholism ..	1	2	12	7
Poor nutrition—chronic alcoholism excluded. (Cases studied shown in parentheses)	5 (43)	4 (17)	3 (25)	4 (35)
Hypertension (over 180 millimetres of mercury, systolic, or 100 millimetres, diastolic)	24	7	15	23

[1] The ages of onset for gastric ulcer, superficial gastritis and gastric ulcer do not differ significantly, but the age of onset for duodenal ulcer is lower. Patients with atrophic gastritis are older when examined at hospital than those with superficial gastritis. These findings suggest that gastric ulcer is more closely related to gastritis than to duodenal ulcer and that atrophic gastritis is a more advanced stage of superficial gastritis.

With the exclusion of all chronic alcoholics about half the patients would usually be classed as nervous, worrying or neurotic (Table I). In many of these, were it not for the biopsy findings, a diagnosis of functional dyspepsia would usually be made. However, it is probable that in this group reassurance and encouragement with domestic difficulties were as useful therapeutically as in cases of functional dyspepsia. But the presence and persistence of the histological changes set two questions: do the personality of the patient and his responses to his environment directly lead to actual mucosal changes of "chronic" character (in the pathological sense), and do the symptoms of fullness, discomfort or nausea arise from altered gastric function due to the local lesion and conditioned by the emotional status? That the latter is more likely is shown in Table II.

Compared with duodenal and gastric ulcer, a family history of dyspepsia was found in surprisingly few cases of gastritis. There were four reports of peptic ulcer and three of dyspepsia of undetermined character. No evidence is available about the presence of other psychosomatic disorders in parents or siblings.

Symptomatology and Examination Findings.

In assessing symptoms we have considered all symptoms of digestive disturbance as being possibly related to chronic gastritis. In particular, if there was dyspepsia with gall-stones and gastritis, we linked the dyspepsia with the gastritis, since no clear demonstration has been made of how cholecystitis or cholelithiasis causes gastric symptoms.

Epigastric Discomfort.

This being borne in mind, there was a history of abdominal pain or discomfort in 31 cases of superficial gastritis and 29 of atrophic gastritis. In 20 and 21 respectively of these the patients were females.

Flatulence was reported in 16 and 30 cases respectively (not entirely coinciding with those in which there was epigastric discomfort).

These were the most important symptoms recorded. The pain was felt in the epigastrium in the majority of cases; but across the upper part of the abdomen, on the right side of the abdomen, behind the sternum and in the lower part of the abdomen were other sites mentioned. It was mild in degree—often being called discomfort, not pain—and rarely became severe. Among the descriptive terms used were "burning", "dull", "full feeling" and "distended". A few patients called it "colicky" or reported a "hungry feeling".

It has already been noted that the presence and severity of symptoms are related to the presence of other manifestations usual in neurosis, and that this may mean that the patient's reaction to the altered function of the stomach is more expressive than that of a well-adjusted person (Table II).

TABLE II.

The Relationship Between Chronic Alcoholism, Psychological Factors and Symptoms in Gastritis.[1]

Diagnosis and Associated Factors.	Symptoms.		
	None.	Mild.	Moderate to Severe.
Superficial Gastritis:			
Chronic alcoholism	10	1	1
Psychological factors (alcoholics excluded)	3	5	17
Remainder	3	2	6
Atrophic gastritis:			
Chronic alcoholism	5	—	2
Psychological factors (alcoholics excluded)	5	9	7
Remainder	25	3	8

[1] Amongst the non-alcoholics it appears that symptoms are more pronounced in patients classified as "nervous", "worrying" or "neurotic".

When the pain is analysed in our series of patients with ulcer there are three features of note: the pain is usually felt in relation to meals, varying in different patients from immediately to three or four hours after meals; the pain is promptly relieved by food and alkaline powder; the pain occurs in bouts separated by intervals of varying length from weeks to years, in which there is complete freedom.

In cases of gastritis many of these features are entirely different. The discomfort associated with gastritis occurs shortly after meals (Table III). This relation is similar with both superficial and atrophic gastritis, and the average interval was found to be half an hour. With gastric ulcer it occurred about one and a half hours and with duodenal ulcer nearly two hours after meals. Pain occurring at night was infrequent with gastritis, but common with ulcer, particularly duodenal ulcer. Of patients suffering from gastritis with dyspepsia a record of the response to food was present for 16. Four stated that food afforded some relief from pain. In these four the pain appeared one hour or more after meals. Thirty patients said that they obtained relief with alkaline powder, while eight said that there was no relief. In cases of gastric and duodenal ulcer there is a more definite history of relief with both food and alkaline powder.

Our impression has always been that the periodicity with gastritis differs from that with ulcer. But on analysis the interval between attacks varies in similar fashion in gastritis and ulcer (Table IV). However, in cases followed up for some time it is evident that patients with gastritis tend to have symptoms for only a few days at a time, whereas in cases of ulcer an attack will last longer.

Diarrhœa.

Diarrhœa was not common, although two-thirds of the patients had achlorhydria. It was present in 10 cases. In one it was associated with diabetes due to hæmochromatosis, in another with ulcerative proctitis and in another with ulcerative colitis. In the remainder the precise mechanism was obscure.

Abdominal Tenderness.

Of patients with superficial gastritis abdominal tenderness was present in 21. It was epigastric or upper abdominal in situation in 14 patients with symptoms. Of the remaining seven, in five the tenderness was adequately explained by other inflammatory conditions. In two it was present without such obvious cause and without symptoms.

In 18 patients with atrophic gastritis there was epigastric tenderness. In only one of these was there present some abdominal condition which could reasonably be the cause of the tenderness.

TABLE III.

The Relation of Discomfort or Pain to Meals in Ulcer and Gastritis.[1]

Time Interval between Meal and Onset of Pain.	Duodenal Ulcer.	Gastric Ulcer.	Gastritis.[2]
Number of cases ..	93	48	60
0 to 1 hour after meals	24 (8)	17 (6)	34 (4)
1½ to 2½ hours	28 (19)	11 (6)	6 (1)
3 to 4 hours	18 (11)	4 (3)	—
Average	1·9 hours	1·4 hours	0·5 hour
No relation to meals ..	19 (5)	11 (2)	14 (4)
No record	4 (0)	5 (0)	6 (1)

[1] Patients with gastritis have either discomfort or pain soon after meals or there is no constant food-pain relationship. In cases of ulcer it is more variable, but usually occurs within 2½ hours of taking food. Even with ulcer there may be no constant food pain relationship. The early onset of pain in some cases of duodenal ulcer is of interest.

Figures in parentheses refer to patients who reported pain at night.

[2] There was no difference between superficial and atrophic gastritis.

Atrophy of the Tongue.

The other physical sign of interest was atrophy of the tongue. When both groups are considered, the tongue was normal in 53 cases, showed slight atrophy in 20, and showed moderate to severe atrophy in 27. This latter group included 16 patients with symptoms. Of the remainder, several had anæmia of one kind or another, a few were chronic alcoholics. One patient with gall-stones and indigestion also had an atrophic tongue due to syphilitic glossitis.

Weight Loss.

Information on the weight loss as stated at the first visit of the patient is shown in Table V, where a comparison with duodenal and gastric ulcer is made. The average in all those cases of gastritis in which some information is available is nine pounds. The group in which there is loss of more than 14 pounds shows no preponderance of superficial or atrophic gastritis.

Nausea and Vomiting.

Nausea as part of the dyspepsia was experienced by eight patients with superficial gastritis and by 11 with atrophic gastritis, but this was not a prominent symptom.

A history of occasional vomiting, usually only once or twice, was obtained in 10 patients with atrophic gastritis and in 11 with superficial gastritis.

Hæmatemesis and Melæna.

Four patients with superficial gastritis and two with atrophic gastritis were admitted to hospital because of hæmatemesis and melæna. A further patient with atrophic gastritis had melæna only. Only one of these had a history of previous hæmorrhage. In no case was the hæmorrhage of great severity, but one patient with atrophic gastritis required blood transfusion. Laparotomy with a view to stopping the hæmorrhage was not performed on any of these patients.

TABLE IV.
The Periodicity of Symptoms in Ulcer and Gastritis.[1]

Disease.	Number of Patients.	Period of Remission.					
		Nil.	Up to One Week.	Up to One Month.	Up to Six Months.	Over Six Months.	No Record.
Duodenal ulcer	93	19	6	11	35	17	15
Gastric ulcer	48	11	2	9	12	6	8
Gastritis	60	13	6	5	17	8	11

[1] Statistical analysis shows no significant differences between the three groups.

Anæmia.

In 23 cases the hæmoglobin value at the time of the first examination of the patient was below 10 grammes *per centum*. In 11 cases this was due to hæmatemesis or melæna or other obvious bleeding. In five there was severe malnutrition associated with chronic alcoholism. In the remaining seven the patients presented to hospital because of their anæmia. The diagnosis in these was microcytic anæmia (two cases), refractory anæmia (two cases), pernicious anæmia (two cases, one doubtful) and hæmolytic anæmia (one case). One patient with microcytic anæmia had a long history of mild diarrhœa and anæmia. Treatment with iron was only partly effective in restoring the hæmoglobin level.

Amongst the patients with hæmoglobin values above 10 grammes *per centum* there were three further cases of pernicious anæmia and one of refractory anæmia.

The result of examination of the fæces for occult blood was negative in the two cases of microcytic anæmia. It was positive in seven other cases in which there was no anæmia.

TABLE V.
The Loss of Weight in Ulcer and Gastritis.[1]

Weight Loss.	Duodenal Ulcer.	Gastric Ulcer.	Gastritis.
Number of cases recorded	78	43	93
No weight loss	39	23	41
Up to seven pounds ..	11	3	15
Seven to fourteen pounds	20	6	18
More than fourteen pounds	8	11	19
Not recorded	15	5	19
Average weight loss ..	6·0 pounds	9·5 pounds	9·0 pounds

[1] There is no significant difference in the three groups.

Peripheral Neuritis.

An absence or decrease in the ankle jerks was found in 24 patients. Of these, 14 had alcoholic peripheral neuritis, three had some central neurological lesion, two were patients over seventy years of age, and one was a diabetic of long standing. The cause of the change in the others was obscure.

Liver Disease.

The liver was palpable below the costal margin in 41 patients. This enlargement was related to fatty liver or cirrhosis of chronic alcoholism in 15 cases, to obstructive jaundice or previous subcostal incision, probably cholecystectomy, in 10 cases, to hepatitis due to causes other than alcoholism in five cases, and to congestive failure and emphysema in one case each. In the majority of the remainder the liver was either just palpable or felt one or two centimetres below the right costal margin. There is nothing to suggest that gastritis *per se* may cause malnutrition with liver enlargement.

X-Ray Appearances.

Barium meal examination of the stomach was carried out on 62 occasions, the patients selected being chiefly those with dyspepsia. In 57 cases the findings were normal. In the others the examination showed pylorospasm (two cases), anatomical abnormality (two cases) and prolapse of gastric mucosa (one case). Except once, when the report was "active", peristalsis was always recorded as "normal". Ptosis, as shown by the finding that the greater curvature was lying well below the level of the iliac crests, was seen in five cases. In no case did the stomach fail to empty itself of barium before six hours. This is in contrast with the view that the stomach with gastritis is low lying and slow emptying (Hurst, 1933).

Gastroscopy Findings.

Gastroscopy was performed in some cases. In cases of superficial gastritis the findings were: normal mucosa, 12 cases; atrophic gastritis, seven cases; exaggerated mucosa folds, one case; active peristalsis, one case. In cases of atrophic gastritis the findings were: normal mucosa, two cases; atrophic gastritis, nine cases; active peristalsis, one case.

Fractional Test Meal Findings.

In all but 12 cases a histamine test meal examination with 0·3 milligramme of histamine was carried out. Aspiration was performed before injection and half-hourly for one and a half or two hours. As much of the gastric contents as possible was removed at each aspiration without posturing the patient. In over half the cases an estimation of peptic activity was made on the specimen removed one hour after histamine administration. The results of test meal examinations (including acid levels in gruel meals) were as follows. Achlorhydria was present in 25 cases of superficial gastritis. The average maximum free acid level in the remainder was 27 units, and for all cases 13 units. The average peptic activity for all cases was 41 units. The range of pepsin in the normal group was 42 to 107 units (Wood *et alii*, 1949*b*).

Achlorhydria was present in 52 cases of atrophic gastritis. The average level of free acid in the remainder was 18 units, or three units for all cases. The average peptic activity was 20 units. The volume secreted in two hours was measured in 11 cases; it ranged from 15 to 153 millilitres, and in seven cases of atrophic gastritis was less than 40 millilitres (range in normal group, 93 to 124 millilitres). No blood was found in the test meal in three-quarters of the examinations.

For comparison an analysis of cases of gastric atrophy (as found with pernicious anæmia) revealed achlorhydria in all cases and an average peptic activity of seven units. (Peptic activity of less than 10 units is not significant by the method used.) The volume of secretion was correspondingly low. The relationship between the histamine test meal result and the gastric mucosa as obtained by gastric biopsy is described elsewhere (Funder and Weiden, 1952; Wood *et alii*, 1949*b*).

Treatment and Management of Gastritis.

If gastritis is associated with some other more serious disease, such as gall-bladder disease or chronic alcoholism with malnutrition, then it is essential to direct treatment mainly towards that associated disease. If no other disease is present, the treatment of the gastritis will be determined by the nature and severity of the symptoms and the patient's attitude towards them. With increase in frequency or severity of the pain, or the presence of vomiting, loss of weight or hæmatemesis, it may be wise or necessary to place the patient in hospital on a full nourishing diet. Alkaline powders may give relief, although theoretically this mode of therapy has no rational basis. We have tried dilute hydrochloric acid in a few patients without much success, and have observed no obvious benefit in patients with diarrhœa. Iron in the form of ferrous sulphate tablets is usually well tolerated and will help to relieve any iron-deficiency anæmia.

In two cases we have found that penicillin given orally, 200,000 units daily in divided doses in milk or as tablets, aids in the resolution of the inflammatory reaction in atrophic gastritis. There was in each case a rapid decrease in the patient's discomfort and a reduction in the cellular infiltration in the series of biopsy fragments. In some cases short courses of oral penicillin therapy have been given in the out-patients' department with apparent benefit.

However, most of the patients are not seriously ill and do not suffer much discomfort, so that they can be treated satisfactorily in the out-patients' department. It is important to see that the patient includes adequate protein and vitamins in the diet. Should any signs of deficiency arise, supplementary vitamins may be given by mouth. Antacid powder may be found useful in some cases, and ferrous sulphate by tablet can be used to correct anæmia. Frequently the patient fears that he has cancer, so that when the diagnosis is established he should be repeatedly reassured that he has an innocent condition. On the other hand, many of them have had minor symptoms for a long time and feel that their story is not fully accepted. Sympathetic attention to their physical complaints, reassurance that they have definite local changes in the stomach to which their symptoms are due, and explanation that emotional factors are important in precipitating further attacks are all of value in increasing the patients' confidence in themselves and the doctor. They are also grateful for a confident prognosis. It can be said that their illness is not serious and is compatible with many years of relatively good health, and that there is no certain evidence that chronic gastritis is a precursor of cancer.

Course of Gastritis.

In 44 cases we have had further information on the course of the pathological lesion, both directly from repeated biopsies and indirectly from test meal examinations. We have found that resolution of the inflammatory process is unusual. More frequently the histological picture is unaltered, or there is a progression from superficial gastritis to atrophic gastritis with a corresponding decrease in the output of acid, pepsin and volume of gastric juice in the test meal examination. The only cases of atrophic gastritis which have resolved completely with return of acid and pepsin secretion have been those induced by X-radiation in the therapy of duodenal ulcer (Doig, Funder and Weiden, 1951).

In none of our cases did we see the development of carcinoma. Nor did we see the progression of the lesion of atrophic gastritis to that of gastric atrophy characteristic of pernicious anæmia (Doig and Wood, 1950). However, in one patient with typical atrophic gastritis, severe atrophy, intestinal metaplasia and minimal cellular infiltration appeared to follow a course of penicillin given by mouth.

In Table VI we have analysed the clinical course in 20 representative cases of patients with symptoms who have been observed on an average of three and a half years (range, two and a half to five and a half years). Their average age when first examined was forty-nine years (range, twenty-six to sixty-four years). However, only one male is included, since symptoms occur more often in females and also since females were more readily available for repeated testing and follow-up. In seven cases there were only mild symptoms while the patients were being followed. In 11 cases dyspepsia of moderate severity was the chief reason for the patients' coming to hospital. In three cases symptoms became much less severe and recurrences infrequent under treatment; in five there was less striking improvement; in two the incidence and severity remained the same; in one symptoms became worse. Two patients first came under observation with hæmatemesis. This was repeated in one patient; in addition, she has multiple complaints, including mild dyspepsia, and has hypertension. The other first came under observation with weight loss, mild hæmatemesis and dyspepsia. Treatment apparently brought about considerable improvement, but there have been subsequent relapses with severe pain and weight loss. These have been such as to warrant admission of the patient to hospital on two occasions.

During the period of observation most patients regained their former weight, 16 gaining an average of seven pounds and four losing an average of five pounds. Most patients felt better as a result of attendance at hospital.

The following two reports (Cases I and II of Table I) describe two typical cases of gastritis.

Superficial Gastritis.—S.L., a married saleswoman, aged thirty-five years, when first examined in 1946, gave a history of eleven years of pain occurring half an hour after meals, relieved by food and alkali, with remissions up to six months. Additional symptoms were a poor appetite, fatigue and mental irritability. Her diet had been moderately satisfactory, although slightly below the optimum requirements. She took a minimal amount of alcohol. She had many domestic worries and was obliged to earn her own living. Examination of the patient revealed a pale intelligent woman with a slightly atrophic tongue and tenderness in the epigastrium. The hæmoglobin value was 11 grammes *per centum*. Barium meal examination findings were negative. A test for occult blood in the stools yielded a negative result. Test meal examination revealed achlorhydria. Treatment consisted of a full diet of high vitamin content, iron tablets, reassurance, together with advice regarding her domestic affairs and the taking of a good annual holiday. Both acid and alkali were tried on occasions with some temporary benefit. In general she preferred the alkali. She paid 24 visits to our follow-up clinic during the five years of observation. On nine occasions her progress was considered to be "good", on seven "fair", and on eight "unsatisfactory"; her main complaints were epigastric pain, lack of appetite and energy, occasional nausea, and worry concerning her domestic troubles. She was greatly benefited by a visit to England. The first gastric biopsy, performed in March, 1949, showed pronounced superficial gastritis, with only slight atrophy of the secreting tubules. (See Figure II of Funder and Weiden's paper published in this issue.) Four subsequent biopsies at regular intervals during the next thirty-two months showed a remarkable uniformity in the histological picture. Five histamine test meal examinations carried out at the same time as the biopsies revealed achlorhydria on four occasions and only eight units of free acid on the other. Pepsin levels (one hour after histamine administration) were all low, being 35, 64, 3, 4 and 6 units.

Atrophic Gastritis.—H.M., a painter, aged forty years, when first examined in the unit in 1949, complained of two years' flatulent dyspepsia, with discomfort coming on immediately after meals and lasting about one hour. There had been one attack of vomiting and diarrhœa and occasional pain in the back. Between the attacks his appetite had been good and his diet moderately satisfactory. He took very little alcohol. On examination he was found to be healthy in appearance, with a good colour. His tongue was slightly atrophic. He had moderate epigastric tenderness. There was no evidence of plumbism. A barium meal examination yielded negative findings. Cholecystography showed no abnormality. The hæmoglobin value was 15 grammes *per centum*. Examination of a blood film produced negative results. For treatment the patient was given acid for nine months with no benefit. There were recurrent attacks of epigastric pain and flatulence lasting from two to four days. He was then given a trial of no treatment and later a course of alkali. His symptoms remained unchanged. During the thirty-one months of observation he paid 15 visits to our follow-up clinic, and his progress was reported to be "good" on five occasions, "fair" on two, and "unsatisfactory" on eight. His main symptoms were similar to his initial complaints. However, he was able to carry on with his work throughout, and his general health remained satisfactory. His weight on first reporting to the clinic was ten stone two pounds, and with improved diet he gained eight pounds in the first six months. His weight then remained constant. Four gastric biopsies were performed at intervals varying from six to twelve months, and they all showed a constant picture of pronounced gastritis with considerable, but not complete, atrophy of the acid and pepsin secreting cells. (See Figure III in Funder and Weiden's paper.) Histamine test meal examinations carried out within a few days of the biopsies all showed achlorhydria and greatly reduced pepsin secretion (eight, nine, nine and six units of pepsin).

Follow-Up.

Our patients are examined at three-monthly to six-monthly intervals, or more frequently if they are in relapse, and we find that they remain moderately well, show little or no apprehension about their illness and, compared with their state of health prior to coming to the unit, they are greatly improved in their general health

TABLE VI. *Summary Presenting Symptoms and Progress in Superficial Gastritis (Cases 1 to 10) and Atrophic Gastritis (Cases 11 to 20) Studied for the Longest Period of Time. (Average: 45 Months.)*[1]

Serial Number, Initials.	Sex, Age (Years).	Presenting Symptoms and other Clinical Features.	Maximum Free Acid.	Progress under Observation.				
				Duration. (Months.)	Weight: Gain or Loss (lbs.)	Dyspepsia.	Other Findings.	Progress.
1 S.L.	F. 35	Eleven years' recurrent epigastric pain of mild to moderate severity.	0	65	+14	Variable course. Occasional epigastric discomfort. Improved by holiday.	See case report.	Fair.
2 F.G.	F. 43	Two months' dyspepsia, debility. Stone in common bile duct, jaundice. Operation.	0	38	+12	Nil since cholecystectomy.	Occasional headache. Blood pressure 210/140.	Good.
3 M.K.	F. 57	Six months' flatulent dyspepsia. Gall-stones. Cholecystectomy. Considerable improvement.	0	50	0	Only minor dyspepsia on infrequent occasions since operation.	Nil.	Good.
4 M.C.	F. 59	Nine years' recurrent mild to moderate epigastric discomfort. Appetite poor.	0	50	+3	Symptoms unchanged. Dyspepsia mostly mild.	Occasional "fainting". Blood pressure 170/110.	Fair.
5 J.B.	F. 34	Fourteen months' mild to moderate epigastric pain. Occasional vomiting. Appetite poor. "Nervy."	20	65	−5	Some improvement with reassurance. Still recurrent dyspepsia.	Nil.	Fair.
6 R.A.	F. 62	Twenty months' mild to moderate epigastric pain immediately after meals. No relief with alkalis. Emotional.	0	47	+11	Still recurrent dyspepsia, lessening in frequency and severity.	Blood pressure 210/120 without symptoms.	Fair.
7 I.F.	F. 43	Nine years' recurrent epigastric discomfort. Partial relief from cholecystectomy eight years ago. Appetite, nutrition poor.	5	30	+6	No change in nature or frequency.	Nil.	Fair.
8 C.C.	F. 37	Four years' recurrent mild to moderate epigastric pain soon after meals increasing in frequency in past four years. Nutrition fair. Moderate beer drinker.	42	34	+1	Recurrent mild to moderate epigastric pain, worse with emotional upsets.	Nil.	Fair.
9 A.N.	F. 64	One week's hæmatemesis. No previous dyspepsia. Probable malnutrition for many years. "Nervy." Blood pressure 208/114.	10	28	+2	One hæmatemesis and melæna of moderate severity. Only slight dyspepsia.	Multiple aches and pains.	Unsatisfactory.
10 G.S.	F. 26	Nurse in X-ray department for eight months. Flatulent dyspepsia half an hour before meals with remissions. Occasional vomiting.	0	30	+6	Occasional mild dyspepsia.	Nil.	Good.
11 H.M.	M. 40	A painter. Two years' recurrent epigastric pain half an hour before meals, lasting one to three days. Remissions of one to two months. One attack of diarrhœa.	0	31	+8	Recurrent attacks of dyspepsia every two to four months of mild to moderate severity. Not prevented by acid or alkali. Well in remission.	See case report.	Fair.
12 K.R.	F. 64	Six years' moderate flatulent dyspepsia with epigastric pain. Long remissions. "Nervy." Pyelitis.	8	47	0	Recurrent mild dyspepsia with remissions of one to six months.	Nil.	Good.
13 D.L.	F. 41	Thirteen years ago hæmatemesis and pyelitis. Since then flatulent dyspepsia with epigastric pain, two to three months per year. Typist. Worries greatly.	0	40	−3	Mostly well, but relapses associated with fenestration operation and domestic overwork.	Otosclerosis (operation).	Fair.
14 B.W.	F. 42	One week's gall-stone colic. No preceding dyspepsia. Cholecystectomy. Routine test showed positive Wassermann reaction. Anti-syphilitic treatment given.	5	56	+11	Well except for occasional mild flatulent dyspepsia.	Nil.	Good.
15 A.W.	F. 56	Twenty years' mild to moderate recurrent epigastric pain half an hour before meals; some relief with powder. Worse last six months. Epigastric tenderness. Nervous disposition.	0	58	+12	Frequent recurrent attacks of epigastric pain, mild to moderate severity half an hour after meals. Some relief from alkali. None from acid.	Essential hypertension. Blood pressure 180/120.	Unsatisfactory.
16 M.W.	F. 64	Thirty years' recurrent mild dyspepsia following prolonged malnutrition in India. Three severe attacks. Laparotomy revealed no abnormality.	0	48	+5	Infrequent mild epigastric pain. Three mild attacks of diarrhœa. Possible benefit from acid therapy.	Hypochromic anæmia. Responded to iron. Coronary occlusion; good recovery.	Fair.
17 H.S.	F. 63	One week's acute cholecystitis with gall-stones. Operation. Post-operative pulmonary collapse. Recovered. No previous dyspepsia.	0	54	+10	Practically nil.	Nil.	Good.
18 M.C.	F. 55	Past year five attacks gall-stone colic. No dyspepsia between attacks. X-ray examination showed gall-stones.	5	41	+7	Remained well after operation. Only slight epigastric discomfort.	Gall-stones removed.	Good.
19 M.H.	F. 62	Six years' recurrent epigastric discomfort, occasional pain immediately after meals. Worse lately. Diet poor. Atrophic tongue and cheilosis.	0	38	−2	General improvement, still recurrent discomfort and three relapses with pain and loss of weight. Improved with bed rest and oral penicillin therapy.	Recurrent cheilosis and atrophic tongue remained. Appetite and diet mostly poor.	Unsatisfactory.
20 I.H.	F. 53	Two years' flatulent dyspepsia, pain immediately after meals; two weeks' recurrent gall-stone colic. Acute cholecystitis, gall-stones. Cholecystectomy.	0	55	−7	Recurrent mild to moderate flatulent dyspepsia every three to six months.	Nil.	Fair.

[1] All patients had characteristic gastric biopsy findings. No evidence of ulcer or gastric carcinoma was found.

						cholecystitis, gall-stones. Cholecystectomy.		

[1] All patients had characteristic gastric biopsy findings. No evidence of ulcer or gastric carcinoma was found.

XUM

and sense of well-being. They eat far more and choose better food. Their weight usually increases at first and then remains stationary at a satisfactory level.

The physician should examine the patient at each visit to assess epigastric tenderness and to make certain that there is no evidence of any associated disease, especially gastric neoplasm. Excess pain or vomiting, progressive loss of weight or anæmia, or a possible epigastric mass immediately calls for a barium meal examination, cholecystography and perhaps gastroscopy, to exclude neoplasm and gall-bladder disease. Should the diagnosis still be uncertain, but the findings highly suggestive of neoplasm, then an exploratory laparotomy must be undertaken. However, we have not as yet observed the development of gastric carcinoma in our series of established cases of chronic gastritis.

In gastritis a relapse in the symptoms is often associated with excess mental stress with its accompanying loss of sleep and impaired appetite. Alleviation of these stresses is of considerable value. The careful taking of a history may lead to sane and helpful advice by the physician. These patients may have difficulty in deciding on and following out a line of action. A wisely timed holiday may be of the greatest help. Many patients in our clinic had not been away from home on holiday for many years. Phenobarbital may be of benefit, especially at night.

The intake of alcohol and its influence on diet should be investigated and adjusted. Alcoholic addicts with symptoms of gastritis as well as other organic lesions, such as alcoholic hepatitis or peripheral neuritis, should abstain from alcohol. "Heavy drinkers" who are healthy except for the symptoms of gastritis are well advised to abstain from alcohol for six months. Then perhaps a moderate intake of alcohol does little harm so long as it is associated with a full diet rich in vitamins. Similarly, for the "light drinker" an occasional beer or a sherry may be of benefit to the tired and worried worker or housewife at the end of a day's toil, so long as the taking of alcohol appears to bear no direct relationship to the pain of gastritis. This direct relationship is seen from time to time, especially with wines such as sherry when they are taken on an empty stomach.

Discussion.

Our study of 112 cases of gastritis over a period ranging up to three years has led us to believe that gastritis is a definite clinical and pathological entity. Although some patients experience neither pain nor discomfort, others do suffer pain varying from mild epigastric discomfort and flatulence to pain of moderate severity. The pain or discomfort may have no constant time relationship to meals; more usually it occurs immediately after meals. Oddly enough, it may be relieved by alkaline powders. In addition to the pain or discomfort there may be hypochromic anæmia, anorexia, occasional vomiting and moderate weight loss. The duration of the symptoms is often many years, with exacerbations and remissions. The physical examination reveals epigastric discomfort and perhaps atrophy of the tongue, test meal examination shows hypochlorhydria or achlorhydria, and X-ray findings are within normal limits. Gastric biopsy of the body mucosa shows varying degrees of inflammatory reaction and atrophy, the term "superficial gastritis" being applied to the finding of inflammatory reaction with little or no atrophy of the acid and pepsin secreting cells, and "atrophic gastritis" to the finding of pronounced inflammatory reaction and atrophy of the essential cells.

The disease pattern in this group of patients—the symptomatology, the physical signs, the test meal examination findings and the gastric biopsy picture—is not constant, but the variation shown is no greater than that found in other conditions, such as peptic ulcer, so that we believe that chronic gastritis is a discrete entity demanding recognition by the physician and surgeon alike.

Our impression is that the previous lack of agreement about the symptomatic importance of gastritis rests on several factors. The early diagnosis of gastritis, as in the writings of Hurst (1933), rested on the presence of excess mucus and the absence of acid in gruel test meal examinations. The error in the use of mucus as a basis has been resolved by the detailed studies of Glass and Boyd (1949) on the various mucus fractions. The results of test meal examinations in which gruel is used have been shown to be easily influenced by temporary nervous inhibition; whereas insulin or histamine can evoke a maximum response which is proportional to the histological appearance of the mucosa (Wood *et alii*, 1949*b*; Funder and Weiden, 1952). Another factor has been the variable interpretation of the gastroscopic appearances; and there is still no unanimity of opinion on this. Part of this variation has been suggested by Wolf and Wolff (1947) to be due to misinterpretation of vascularity and other aspects of the increased or decreased gastric activity. Lastly, based on minute separation of gastroscopic appearances, a detailed analysis of symptoms has led Palmer and his colleagues to the conclusion that gastritis cannot be related to symptoms (Findley *et alii*, 1949; Ricketts *et alii*, 1949). However, we have shown that, when considered as a group, patients with a histological diagnosis of gastritis show a recognizable though variable and usually mild clinical picture.

Two clear ætiological factors are known for gastritis. First, it may develop from X-radiation, either as superficial gastritis or as atrophic gastritis (Doig, Funder and Weiden, 1951; Ricketts *et alii*, 1948). Here there is no difference in the intensity of radiation at the surface when compared with the depths. Second, gastritis may follow chronic alcoholism, although even here the action may be an indirect one through the accompanying malnutrition rather than by the direct effect of alcohol. Since both these agents affect the whole depth of the mucosa, and since surface agents have not been implicated, the main emphasis must be on alteration in the *milieu* of the mucosa determined by circulating toxins, alterations in blood supply, or nervous influences. Such an hypothesis would permit inclusion of psychic stress as a factor in the actual genesis of the lesion or its persistence. However, the data suggest that the most important action of psychic stress is to render the patient sensitive to painful stimuli.

The mechanism by which symptoms arise is not clear. Mourao and Schindler (1949) have suggestive evidence that it is due to a disturbance of the "tonus" mechanism of the stomach—that is, the mechanism by which the stomach relaxes to accommodate an increased content. Such a mechanism is in keeping with the occurrence of pain soon after eating and would be influenced greatly by the emotional state of the patient.

With regard to the course of the disease, our studies suggest that superficial gastritis may heal with the disappearance of symptoms and return of the test meal findings to normal, but far more often the disease remains or progresses to atrophic gastritis with persistent hypochlorhydria or achlorhydria, reduced pepsin concentration and low total volume of secretion. The only cases of severe atrophic gastritis in which healing occurred with return of acid and pepsin were cases of duodenal ulcer in which the atrophic gastritis and hypochlorhydria had been produced as a means of therapy (Doig and Funder, 1951).

Although the work of Comfort, Kelsey and Berkman (1947) suggests that achlorhydria, and therefore probably gastritis, precede the development of cancer by many years in some cases, we have no evidence to support or refute this. Perhaps further studies may throw some light on this problem.

We did not see the development of the picture of gastric atrophy of pernicious anæmia except in one case under special circumstances. Our opinion on the evidence we have is that the lesion of atrophic gastritis does not show progression to gastric atrophy (Doig and Wood, 1950; Doig *et alii*, 1950; Motteram, 1951).

With regard to the gastric atrophy of pernicious anæmia, we do not consider that cases of atrophic gastritis progress to gastric atrophy with the picture of severe atrophy, intestinal metaplasia and relative absence of inflammatory changes (Doig and Wood, 1950; Doig *et alii*, 1950; Motteram, 1951). However, the available evidence for this opinion is rather meagre.

The relationship between gastritis and peptic ulcer is of importance. In our series the symptoms of gastritis (both superficial and atrophic) and gastric ulcer most commonly

begin in the fifth decade of life, whereas those of duodenal ulcer usually appear earlier, in the third or fourth decade. It is possible that early patchy superficial gastritis may bear some relationship to the genesis of acute gastric ulceration. Should the gastritis become widespread and more pronounced, the reduction of the secretion of acid and pepsin would lessen the tendency to ulceration. Our observations do not add to our knowledge of the cause of ulcer, and more evidence is needed to support this hypothesis.

Summary.

1. A series of 112 cases of diffuse gastritis (48 cases of superficial gastritis and 64 cases of atrophic gastritis) has been reviewed with regard to ætiology, symptomatology, diagnosis, prognosis and management, and compared with a similar series of cases of gastric and duodenal ulcer.

2. The diagnosis has been made from the history, physical examination and test meal examination findings and histological appearance of gastric biopsy fragments obtained by the flexible gastric biopsy tube designed in this unit.

3. No constant ætiological factor was found. The most important factor was chronic alcoholism with malnutrition. About half the patients would usually be classed as nervous, worrying or neurotic. The evidence favoured the idea that this was related to the actual appearance of symptoms, although a mechanism has been suggested which could introduce psychic stress as a causal factor.

4. The sex distribution for superficial gastritis was 12 males and 36 females; for atrophic gastritis it was 28 males and 36 females.

5. The age distribution (age of onset of symptoms and age when patient was examined at hospital), when compared with that of duodenal and gastric ulcer, shows that patients with duodenal ulcer develop symptoms and report to hospital at an earlier age than those with gastric ulcer, superficial gastritis and atrophic gastritis. The longer history and greater age of patients with atrophic gastritis is in agreement with the view that this is a further development of superficial gastritis.

6. The chief symptoms were epigastric pain or discomfort (in 60 cases) and flatulence (in 46 cases). The pain was usually mild and occurred on an average of half an hour after meals. Relief with food and alkaline powder was inconstant. In cases of ulcer the pain was of greater intensity, occurred about two hours after meals, and relief with food or powder was more constant. The intervals of freedom varied in both cases similarly, but in cases of gastritis the bouts were of shorter duration.

7. Other features of gastritis were abdominal tenderness, usually epigastric, atrophy of the tongue, loss of weight and anæmia. The anæmia was contributed to in part by malnutrition and hæmatemesis. Diarrhœa occurred in only 10 cases.

8. Histamine test meal examination showed that there was a pronounced reduction in the acid and pepsin secretion. Achlorhydria was present in 25 cases of superficial gastritis and in 52 cases of atrophic gastritis.

9. Treatment, apart from that of associated lesions, was to ensure a full diet and to reassure the patient about the nature of the disease. It was not successful in effecting a return of the mucosa to normal or a return to normal of the test meal findings. Support, sedation and help in relieving physical and mental stress were useful in preventing relapses.

10. Gastritis is a common disease and may be a cause of continued ill health in the patient. Its exacerbations enter into the differential diagnosis of hæmatemesis and carcinoma of the stomach. It is worthy of further study in relation to cancer and pernicious anæmia.

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THE CORRELATION BETWEEN TEST MEAL FINDINGS AND THE HISTOLOGY OF THE STOMACH AS SHOWN BY GASTRIC BIOPSY.[1]

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IN a previous paper (Wood *et alii*, 1949) an analysis of 54 cases indicated that there is a relation between the gastric secretion (volume, acid and pepsin) and the histological appearance of the mucosa of the body of the stomach. In that series no attempt was made to group the patients in terms of the severity of their mucosal lesion. Now 268 patients with a normal mucosa or a diffuse gastric lesion have been examined by means of a histamine test meal and by gastric biopsy with the flexible gastric biopsy tube designed in our unit. All patients

[1] Aided by a grant from the National Health and Medical Research Council of Australia.

were subjected to a careful clinical examination and, when indicated, X-ray examination. Gastroscopy and laparotomy were performed in a few instances. Patients with local gastric lesions, namely, gastric ulcer and carcinoma, were excluded from the present series.

Methods.

By the technique described (Wood *et alii*, 1949) fragments of mucosa were obtained from the body of the stomach and fixed in 10% formol-saline solution. Paraffin sections were stained with hæmatoxylin and eosin, hæmatoxylin and mucicarmine, and a trichrome stain (methyl violet-orange G, Ponceau-phloxine and methyl green). Details of the use of the last-named stain have been described elsewhere (Motteram, 1951).

The test meals were performed by aspirating the fasting gastric contents, injecting 0·3 milligramme of histamine hydrochloride subcutaneously, and withdrawing three further specimens of gastric juice at half-hourly intervals. Topfer's reagent was used as an indicator. Hypochlorhydria was considered to be present when less than 20 units *per centum* of free hydrochloric acid were secreted. In a selected group of patients pepsin concentration was

TABLE I.
Relation between Histological Classification of Gastric Mucosa and Hydrochloric Acid Secretion.

Histological Classification.	Number of Cases.	Average Age. (Years.)	Hydrochloric Acid Secretion. (Units.)			
			>60	20 to 59	0 to 19	0
Normal	139	44	57	68	10	4
(i) Duodenal ulcer sub-group ..	63	46	35	27	1	0
(ii) Non-ulcer sub-group	76	43	22	41	9	4
Superficial gastritis	44	49	2	10	9	23
Atrophic gastritis	56	57	0	3	7	46
Gastric atrophy	29	60	0	0	0	29

estimated at the end of one hour by the method of Anson (1938), a hæmoglobin substrate being used. The pepsin was expressed as units, a unit being the amount of enzyme which liberates one milliequivalent of tyrosine from the hæmoglobin substrate in ten minutes at 25° C. The normal range was considered to be 30 to 70 $\times 10^{-4}$ units. Pepsin was not considered present when a value of less than 10×10^{-4} units was obtained.

Histological Classification and Clinical States.

By means of the gastric biopsy it is now possible to distinguish normal gastric mucosa and three abnormal lesions of the mucosa: superficial gastritis, atrophic gastritis and gastric atrophy (Doig and Motteram, 1950; Motteram, 1951).

These states are illustrated in Figures I to IV and may be described briefly as follows.

Normal Gastric Mucosa (*Figure I*).—The surface epithelium of the gastric ridges and pits is tall columnar and regular. The tubular gastric glands fill the *lamina propria* and consist of parietal (acid-secreting) and chief (pepsinogen-secreting) cells. The chief cells are more numerous towards the base of the glands. A few mucus cells may be present at the neck of the glands. Only a few wandering cells, large and small mononuclear cells and plasma cells, are present in the *lamina propria*.

Superficial Gastritis (*Figure II*).—The surface epithelium of the gastric ridges and pits shows varying degrees of irregularity and loss of height. The *lamina propria* has an excess of wandering cells, maximal in the superficial portion between the gastric pits. The wandering cells present in excess include polymorphonuclear leucocytes, plasma cells and small and large mononuclear cells. The gastric tubules show no gross change, though chief and parietal cells may be slightly reduced in number and a greater proportion of the tubular epithelial cells are mucus-secreting than in the normal mucosa.

Atrophic Gastritis (*Figure III*).—Changes in the surface epithelium and accumulation of wandering cells in the *lamina propria* are similar to those seen in superficial gastritis. Occasionally metaplasia of the surface epithelium to that of an intestinal type is present. In addition the gastric tubules show changes often gross in degree. There is a reduction in

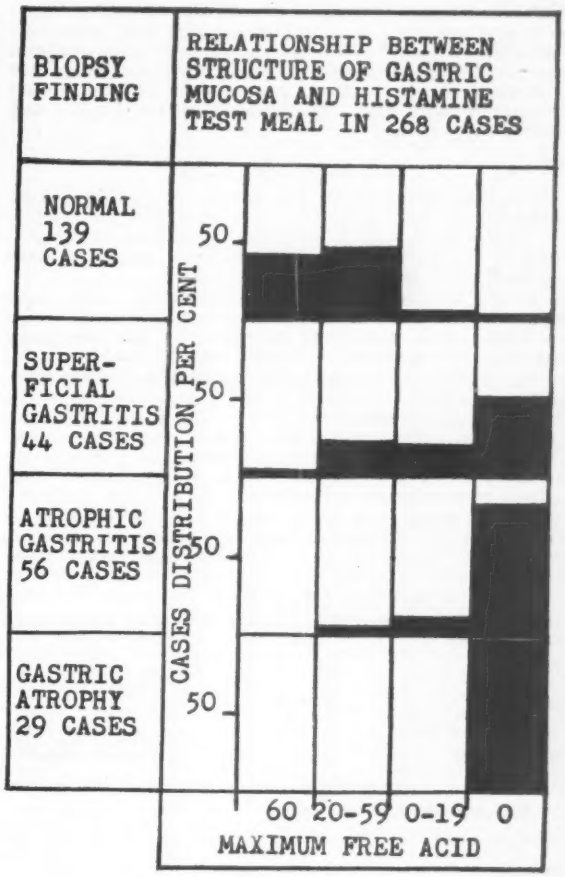

FIGURE V.
Showing that there is a close relationship between the histology of fragments of mucosa obtained from the body of the stomach by biopsy and secretion of acid after histamine stimulation.

the number of tubules and their arrangement in the *lamina propria* is irregular. Chief and parietal cells are greatly reduced in number. Often large stretches of tubular epithelium stain faintly with mucicarmine and fail to stain specifically for acid or pepsinogen. The mucosa is often reduced in depth.

Gastric Atrophy (*Figure IV*).—There is constant reduction in depth of the mucosa. The surface epithelium is for the most part tall columnar and regular, as in the normal mucosa. Metaplasia of the surface epithelium and gastric pits to that of the intestinal type of mucosa is frequent and often considerable in extent. The gastric pits extend almost to the *muscularis mucosæ*. The few tubular glands which may be present stain with mucicarmine. Chief or parietal cells are rarely seen. Wandering cell accumulation in the *lamina propria* is inconspicuous, in contrast to the considerable cellular exudate of atrophic gastritis.

Included in the patients examined in this series were patients with duodenal ulcer and with pernicious anæmia and/or subacute combined degeneration of the spinal cord.

Our experience has shown that in patients with duodenal ulcer the mucosa of the body of the stomach is usually normal. Gastric atrophy is the typical gastric lesion of pernicious anæmia and/or subacute combined degeneration of the cord (Doig, Motteram, Robertson and Wood, 1951).

The almost invariable similarity of fragments taken at the same biopsy operation or of serial biopsy specimens taken at short intervals in the same patient has led us to believe that the lesions of superficial gastritis, atrophic gastritis and gastric atrophy are diffuse and uniform.

The 268 patients examined included 29 with pernicious anæmia, who had gastric atrophy, and 63 with duodenal ulcer, who had a normal mucosa. The remaining 176 comprised patients suffering from a wide range of clinical conditions, with some bias towards gastric, hepatic and gall-bladder disease.

Results and Discussion.

The distribution of the 268 samples of gastric mucosa between the four histological categories and the relation to hydrochloric acid secretion are presented in Table I and in Figure V. Table I also includes the average age of patients in each group.

It will be seen that there is a considerable depression of acid secretion associated with superficial gastritis and a still further depression with atrophic gastritis. All patients with gastric atrophy had achlorhydria.

The depression of acid secretion associated with superficial gastritis is of interest in that abundant parietal cells of normal appearance were present. The failure to secrete normal amounts of acid is to be sought, therefore, in some disorder of function associated with the inflammatory exudate or in a structural change not demonstrable by ordinary histological methods.

Pepsin estimations were carried out in a group of patients with achlorhydria, and the findings are shown in Table II. While the observations are insufficient to be conclusive, they suggest that there is a progressive reduction of pepsin secretion with increasing deviation from the normal histological appearance of the mucosa, and that depression of pepsin secretion lags somewhat behind that of acid secretion. Some support, on morphological grounds, for the latter point is afforded by our observation that in atrophic mucosa in which no demonstrable parietal cells are present small groups of chief cells at the base of the gastric glands are sometimes found.

TABLE II.
Pepsin Secretion in a Group of Patients with Achlorhydria and Abnormal Gastric Mucosa.

Histological Classification.	Number of Cases.	Pepsin Secretion. (Units × 10^{-4}.)	
		<10	>10 (Average value shown in parentheses.)
Superficial gastritis	16	4	12 (50)
Atrophic gastritis	32	12	20 (28)
Gastric atrophy	12	7	5 (13)

The finding of a significant degree of pepsin secretion in five of the twelve cases of gastric atrophy associated with pernicious anæmia indicates the error of the view that pepsin secretion is invariably absent in this condition.

Summary.

1. Gastric biopsy and histamine test meal examination have been carried out on a series of 268 patients.

2. The lesions of superficial gastritis and atrophic gastritis were found to be associated with a depression of hydrochloric acid secretion, more severe with atrophic gastritis than with superficial gastritis.

3. All patients with gastric atrophy (those with pernicious anæmia and/or subacute combined degeneration of the spinal cord) had achlorhydria.

4. Suggestive evidence is presented of a similar but less profound depression of pepsin secretion with increasing deviation from the normal mucosa.

5. Pepsin secretion has been demonstrated in some patients with pernicious anæmia.

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GASTRIC AND DUODENAL ULCER: A SURVEY OF 141 CASES.[1]

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THERE is evidence to show that the relative incidence of gastric and duodenal ulcer varies in different parts of the world. Such variations appear to depend on a changing incidence of gastric ulcer, and they lend weight to the contention that the two diseases, although sharing many features, have fundamental differences. The intensive survey carried out by Doll and Avery Jones (1951) in England will undoubtedly shed much light on the detailed incidence of peptic ulcer in a population, and from similar surveys in other countries differences may appear which suggest important ætiological factors. Such a survey is a vast undertaking. We have in this paper made a limited study of a group of hospital patients.

Our aim is to record the features of gastric and duodenal ulcer as found in hospital out-patient and in-patient practice in Melbourne. This study was also made to serve as a control to a group of cases of chronic gastritis investigated under similar conditions (Doig and Wood, 1952).

Method of Study.

The patients consisted of those first examined during the period June, 1946, to November, 1950. Only those who have been followed up for a reasonable period have been included. There were 93 patients with duodenal ulcer (75 males and 18 females) and 48 with gastric ulcer (32 males and 16 females).

Diagnosis was based chiefly on radiological and operation findings. In 74 cases diagnosis of duodenal ulcer was based on deformity of the cap; in 18 there was also a crater.

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Of 12 cases in which there was no deformity of the cap, a crater was seen in five and diagnosis was confirmed at operation in two. Diagnosis of duodenal ulcer in the remaining five cases seemed reasonable on clinical grounds with lesser radiological findings. In seven cases the diagnosis was "pyloric obstruction", and in one the diagnosis was made at autopsy, sudden death having occurred from melæna.

With regard to the 48 cases of gastric ulcer, in 33 cases X-ray examination revealed a crater in the stomach. In 12 cases no crater was seen, but the diagnosis was based on findings at operation in four cases and at gastroscopy in two, on clinical findings and less definite radiological changes in five and on autopsy findings in one.

Three patients with duodenal ulcer had also had evidence of gastric ulcer at some time; they have been included in the duodenal ulcer group. In the same period five cases of anastomotic ulcer were found. These have been excluded from the survey.

The cases were studied in regard to clinical history and findings on physical examination, and on results of barium meal examination, fractional test meal examination and gastroscopy. Laparotomy was performed in 45 cases.

Age of Onset.

If previous indigestion recorded by the patient is accepted as being due to ulcer, then the average age of onset of symptoms was thirty-six years for duodenal ulcer and forty-four years for gastric ulcer. However, the patient did not come to the hospital until some nine years later in cases of duodenal ulcer and seven years later in cases of gastric ulcer—these were the averages for intervals which ranged from one month to thirty years and from two months to twenty-three years respectively.

The interval may be relatively symptom-free or contain major episodes, such as hæmorrhage or perforation. Attendance at hospital as a rule means some increase in the severity of the disease. There is therefore the impression that numbers of patients with ulcer respond to simple measures of treatment and are never included in any hospital figures.

Family History.

Inquiry concerning dyspepsia in relatives showed a difference between duodenal and gastric ulcer. In the families of patients with duodenal ulcer there were six cases of duodenal ulcer, two of gastric ulcer, six of "ulcer" and eleven of dyspepsia, in father or mother, brothers or sisters. For gastric ulcer the figures were 0, 1, 2, 0 respectively.

Social and Personal Factors.

Of the 93 patients with duodenal ulcer only three had positions involving trust or responsibility. Only three men (other than managers) were workers on their own account; their occupations were dairy farmer, milk carter and tailor. Amongst the men several had subnormal intelligence. We present these facts since the undoubted high incidence of duodenal ulcer in executives has perhaps blinded us, so that we regard a tense, driving personality as almost essential to the diagnosis. Although this was found in many patients it was by no means universal. Often their occupations were not associated with responsible positions.

With regard to gastric ulcer, it was evident that many patients showed emotional factors of note, but no real difference could be made out between the types seen in duodenal and in gastric ulcer, despite the obvious differences between the two diseases in other directions.

Symptomatology and Examination Findings.

Neither the gastric nor the duodenal ulcer patients presented any unusual features in regard to symptoms. In the majority of cases there was a preceding history of indigestion before the present attendance at hospital. However, in eight cases of duodenal ulcer and in six of gastric ulcer the initial symptom was not a pain but epigastric discomfort, heartburn or waterbrash. Bleeding was the first manifestation of three duodenal ulcers and of one gastric ulcer. Vomiting, loss of weight, or loss of energy occurred in four cases of duodenal ulcer and weakness in one case of gastric ulcer. In only one instance was it recorded that the patient's first symptom was perforation. This was a case of duodenal ulcer which perforated when the man was aged thirty-nine years. Apart from these exceptions, pain was the first symptom in 117 of the 141 cases (83%).

It is of course difficult to detect large numbers of patients with an ulcer but no symptoms: all of our patients have had symptoms of active ulceration. This is not so in gastritis, in which the test meal examination can select cases more or less independently of symptoms. This is an unavoidable difference between the two groups of cases we are studying.

Pain.

Pain was referred usually to the epigastrium. It may also be felt behind the sternum, under the right or left costal margin, in the umbilical area or in the right iliac fossa or the back. These variations were more usual with gastric ulcer, except that severe pain, felt in the lumbar region, was a useful diagnostic feature of penetrating duodenal ulcer. The descriptive terms used in cases of duodenal ulcer were "burning", "gnawing", "dull", "nagging"; in gastric ulcer "burning" and "colicky". The pain averaged from mild to moderate severity in both gastric and duodenal ulcer. But in nine cases of duodenal ulcer it was recorded as "severe". All these patients were operated on. Eight males had an elective partial gastrectomy. In one female, operation was performed because of a mistaken diagnosis of acute cholecystitis. The ulcer was oversewn and then the patient was treated medically.

The pain was related to meals in all but 23 cases of duodenal ulcer and 16 cases of gastric ulcer. The average interval between partaking of food and the onset of pain was two hours for duodenal ulcer and one and a half hours for gastric ulcer, but in both groups it varied from immediately after eating to about four hours after. Pain at night was more likely to be present when a long interval between eating and onset of pain was present.

Relief of pain from the taking of food was said to occur 48 times in duodenal ulcer and 32 times in gastric ulcer, and not to occur nine and six times respectively. Alkaline powder was also effective 72 times in duodenal ulcer and 26 times in gastric ulcer, and was not effective five and seven times respectively. The corresponding figures in gastritis were relief from food in four cases and no relief in 12, and from alkaline powder in 30 cases of 38. Although no routine inquiry was made, we feel that in ulcer it is more usual to note complete relief in about five minutes, while in gastritis the relief after the taking of alkaline powder may be incomplete and takes longer to become evident.

The periodicity is similar in the two varieties of ulcer and in gastritis, although prior to this survey we felt that some difference existed. It is impossible to give any reasonable average interval, since so often the statement is made that it is "months", or it varies so widely. In Table IV of our accompanying paper (Doig and Wood, 1952) there is no significant difference shown between the three groups of cases.

Hæmatemesis and Melæna.

Hæmatemesis and melæna were frequent complications of ulcer, but it seemed that bleeding carried a different significance in the natural history of the two diseases. In duodenal ulcer it was recorded at least once in the histories of 40 patients. In age they did not differ from the remainder of the patients. The average age at the first hæmorrhage was forty years. In 22 cases the bleeding had occurred in the past, while in 15 the hæmatemesis or melæna was the reason for coming to hospital. Three patients had bled since coming under attention. This group of patients, although less than half the total, includes half of the patients who suffered perforation and 14 of the 27 who had elective operations. It is probable, therefore, that perforation, bleeding and failure to do well on medical treatment are all reflections of an intractable course. Of these 40

patients who had hæmatemesis or melæna, one died of pulmonary embolus ten days after a severe hæmatemesis; one on whom elective gastrectomy was carried out for repeated hæmatemesis died five days after operation from aortic stenosis; the remainder recovered satisfactorily from their bleeding.

In the group of gastric ulcer patients, 10 gave a previous history of hæmatemesis or melæna and 10 came under observation for the first time because of hæmorrhage. In gastric ulcer the patients who bled again showed no difference in age of onset or age at first hospital visit. Bleeding occurred at the age of about forty-eight years on the average, although the ages ranged from nineteen to sixty-eight years.

In neither group did hypertension have any relation to bleeding.

Perforation.

Fourteen patients with duodenal ulcer had a history of previous perforation. Two patients were admitted with perforated duodenal ulcers after attending previously because of other symptoms. A third patient was admitted because of hæmatemesis, suffered a perforation the day following admission, and later required partial gastrectomy because of obstruction. The average age of the patients at the time of perforation of a duodenal ulcer was forty-four years, but these patients did not come under our care till an average of seven years later. In nine of the 14 cases a past history of perforation accompanied a decision to operate. Management of the remainder by medical treatment has been satisfactory.

In gastric ulcer, six patients gave a history of perforation; five of these have had partial gastrectomy; one had a hæmatemesis, followed by perforation, after being under observation for four years. The average age of onset of this group was forty-four years. Perforation occurred between the ages of forty-three and sixty-five years—a later age group with a much wider spread than in duodenal ulcer.

Abdominal Tenderness.

Tenderness in the epigastrium or above and to the right of the umbilicus was recorded in 47 cases of duodenal ulcer. It varied considerably in degree. It was noted in 25 cases of gastric ulcer.

Weight Loss.

The average weight loss in 133 cases studied was six pounds for duodenal ulcer and ten pounds for gastric ulcer. In both groups half the patients reported no weight loss in the period prior to their coming to hospital. Weight loss greater than fourteen pounds occurred more frequently with gastric ulcer. Disturbance of appetite occurred with duodenal and gastric ulcer, but grading of the appetite did not run parallel with weight loss.

Gastroscopic Findings.

Gastroscopy was performed in 28 cases of gastric ulcer and in 39 cases of duodenal ulcer. In gastric ulcer it was of value in locating an ulcer which was not visualized or was doubtful in the X-ray examination, and in differentiating between simple ulcer and carcinoma. It is of limited value in the diagnosis of gastritis (Doig and Wood, 1952). We have used the Schindler gastroscope, but we are confident that better results can be obtained with the Herman Taylor gastroscope. We are grateful to Dr. W. E. King for his help with the Herman Taylor instrument in some of our cases.

In the 28 cases of gastric ulcer we were able to see the ulcer in 10 and scarring suggestive of a healed ulcer in three. The remainder showed no abnormality.

Gastroscopic examination of 39 patients with duodenal ulcer showed a normal stomach in 35. The remaining four had some evidence of antral gastritis.

We have had limited but encouraging experience in the early diagnosis of bleeding gastric ulcer as advocated by Avery Jones (1947). With full sedation our patients have withstood the examination very well and suffered no after effects.

Test Meal Findings.

Funder and Weiden (1952) have enlarged on our earlier study (Wood *et alii*, 1949) on the relation between test meal findings and biopsy results by a comparison between a normal group of patients and those with duodenal ulcer, superficial gastritis, atrophic gastritis and gastric atrophy. It has been found that patients with duodenal ulcer have a typically high concentration of free acid and a large volume of secretion, whereas those with gastric ulcer have a somewhat variable concentration and volume although these are mostly within the normal range; the figures for patients with superficial gastritis, atrophic gastritis and gastric atrophy are in descending order of magnitude. (See Figure V in the paper by Funder and Weiden, 1952.)

Our interpretation of these findings is that in duodenal ulcer there is a normal gastric mucosa capable of responding actively to a standard stimulus of histamine, whereas in gastric ulcer there is partial depletion of the normal acid-secreting mucosa due to the ulceration with a surrounding zone of gastritis; in the diffuse lesions of superficial gastritis and atrophic gastritis there is inflammatory reaction with increasing atrophy of the gastric mucosa. In gastric atrophy there is extreme atrophy with little or no inflammatory reaction. Thus these changes in secretion can be explained, at least in part, on anatomical grounds. Why the patient with duodenal ulcer has hyperchlorhydria cannot be explained on anatomical grounds, according to our observations on gastric biopsy fragments. On our evidence we cannot agree with the findings of Tongen (1950), who suggests that there is actual hypertrophy of the gastric mucosa in hyperchlorhydria.

Treatment.

Hospital Management.

In the management of a patient with a duodenal or a gastric ulcer, the physician has two problems: firstly, should the patient be admitted to hospital for the initial period of treatment? and secondly, is operation indicated? If the patient has definite or threatened complications, such as perforation, bleeding (be it acute or chronic), obstruction, penetration with severe unremitting pain especially in the back or possible neoplastic changes, the decision is a simple one—admission to hospital is essential. But we also feel that when the diagnosis of gastric or duodenal ulcer appears to be definite it is always wise to begin treatment with four to six weeks in hospital. In this series we have admitted 24 patients at the time the diagnosis was made by us, apart from 86 who had the complications listed above. The patient is often resistant to this advice, since he does not feel very ill and will be absent from work; but our experience suggests that it is the wisest course to take.

In hospital treatment includes antacids, atropine, sedatives and a liberal diet taken in three meals daily interspaced with milk drinks to help buffer acid secretion. We have not had experience with the use of "Banthine" (G. D. Searle and Company, Chicago). The response to these measures is usually most satisfactory. The pain ceases, the patient becomes greatly encouraged and confident, and he can usually return to his business and social life with renewed vigour and with a more rational appreciation of an orderly way of life.

If there is more severe pain, which is slow to respond, or if there is pyloric obstruction, or if relapse has led to impaired appetite or loss of weight, continuous intragastric drip therapy has been of value (Garlick, 1949). We have used this for 20 patients, 14 of whom have come to operation. It relieves the severe pain in penetrating ulcer, and also improves the nutrition of those patients with obstruction and is especially valuable pre-operatively (Grayton Brown, 1951).

Out-Patient Treatment.

In the out-patient department treatment is planned according to the home life, occupation and mental make-up of the patient. All patients require three main meals per

ILLUSTRATIONS TO THE ARTICLE BY J. F. FUNDER AND S. WEIDEN.

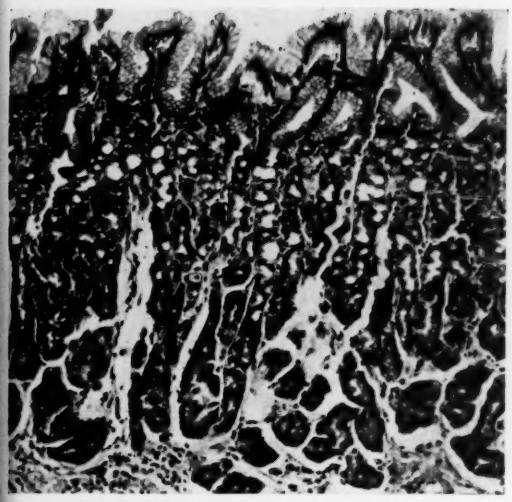


FIGURE I.

Normal gastric mucosa obtained by biopsy. Test meal findings: normal. Hæmatoxylin and eosin stain, × 130.

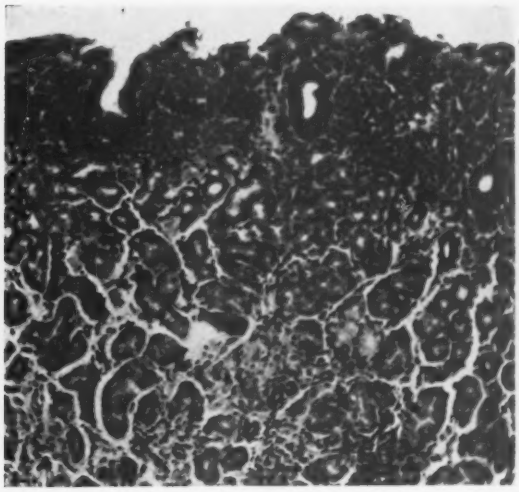


FIGURE II.

Gastric mucosa obtained by biopsy showing superficial gastritis. Test meal findings: achlorhydria. Hæmatoxylin and eosin stain, × 130.

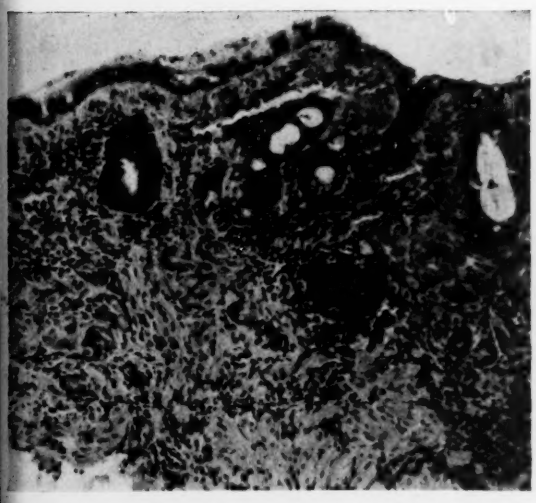


FIGURE III.

Gastric mucosa obtained by biopsy showing atrophic gastritis. Test meal findings: achlorhydria. Hæmatoxylin and eosin stain, × 130.

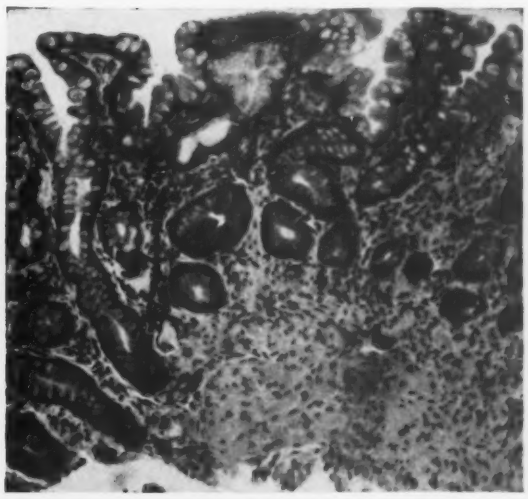
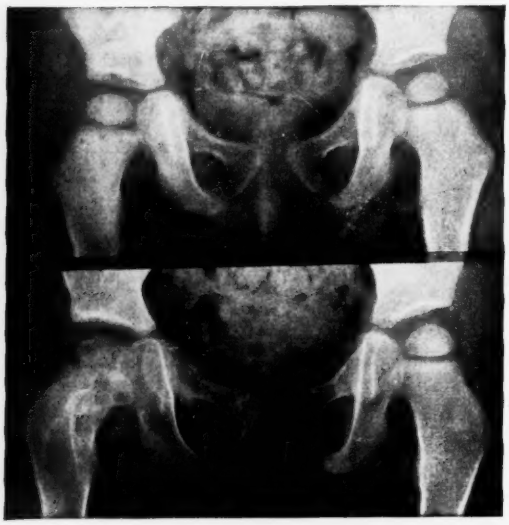


FIGURE IV.

Gastric mucosa obtained by biopsy showing gastric atrophy in a case of pernicious anæmia. Test meal findings: achlorhydria. Hæmatoxylin and eosin stain, × 130.

XUM

ILLUSTRATIONS TO THE ARTICLE BY BRIAN E. FRECKER.



FIGURES Ia AND Ib.

FIGURE II.

FIGURE III.

FIGURE IV.

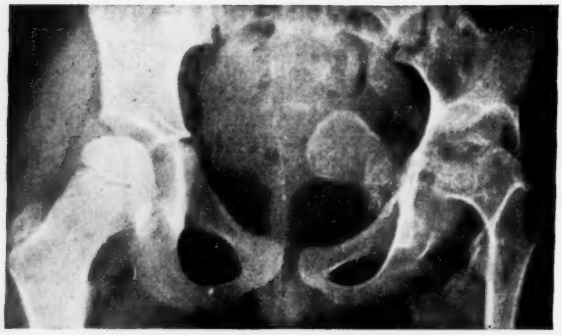


FIGURE V.

FIGURE VI.

day interspaced with three drinks of milk or milk-containing drinks to buffer the secretion of acid. We have favoured magnesium trisilicate as an antacid, giving it after the main meals and at night. However, some patients obtain better relief with "Amphogel" (Wyeth Incorporated) or the standard "pulv McLean A". One patient who took sodium bicarbonate to excess came to hospital with a penetrating ulcer and the symptoms of alkalosis. But if the powders are not taken to excess and there is no vomiting, there is no risk of alkalosis. Belladonna mixture is valuable for the first three months for highly active patients with spasmodic pain and high secretions, as for some patients with duodenal ulcer. Sedatives, in particular phenobarbital, we have used freely, and in some cases we have pushed the dose up to two grains during the day and two grains at night. But it is important not to render the patient weak and miserable because of overdosage.

The patients should be examined from time to time by the physician. Our practice has not been rigid, but we usually examined them at monthly intervals for several visits, and then less frequently over the next year. If they were doing well we then examined them at three-monthly intervals for the second year and thereafter six-monthly. In addition to the symptoms, the signs yielding most information were the presence of abdominal tenderness or mass, anæmia or occult blood in the stools, and change in weight. Slow loss of weight might accompany penetration, obstruction or the development of a gastric carcinoma. However, in none of our cases of gastric ulcer did we see the development of a carcinoma in this manner.

The physician should discuss with the patient how treatment is suiting his way of life; it should suit his home life, his work and his social life without imposing any undue stress. Over-treatment can be most arduous to the patient; he should be happy and pleased with his improvement. We have aimed at abstinence from alcohol and tobacco for six months, and thereafter their use in moderation and with meals or after meals. Theoretically they may aggravate an ulcer, but we have no evidence in our series that this is the case.

Treatment of Hæmorrhage.

Our practice in the management of hæmatemesis and melæna is along the "modern" conservative lines with early liberal feeding and the free use of blood transfusions. The results of the measures adopted do not seem to be at variance with the views on prognosis expressed by Avery Jones (1947) or in the report of Lawson on a series of cases from the Royal Melbourne Hospital in 1945. To achieve early diagnosis we have tried both gastroscopic and X-ray examination without harm to the patient (Avery Jones, 1947). Recently, to replenish the depleted iron stores at an early stage, Dr. Joske of our unit has tried the addition of 600 milligrammes of iron, after a test dose, to the transfusion. In the more remote management of anæmia resulting from an acute hæmorrhage or from chronic loss we have found that ferrous sulphate or ferric ammonium citrate is usually adequate.

In this series of cases we have been responsible for the management of 28 patients with acute hæmatemesis or melæna. None of these patients died as a result of bleeding in the case of gastric ulcer and only one patient with duodenal ulcer died from this cause. Only one patient has been operated upon at the time of a severe hæmorrhage. Although no general indication for operation can be laid down, we have taken the following as guiding suggestions. Should a third hæmorrhage occur after adequate resuscitation has been performed twice, all within three weeks, then surgery is indicated, and for ulcer the operation of choice is partial gastrectomy. For the younger patient greater conservatism is shown; for the older patient, the more severe the hæmorrhage, and the worse the past history of symptoms, the earlier is surgical interference indicated. With the use of rational resuscitative methods from the beginning, more skilful surgery, improved anæsthetic methods and the use of antibiotics, the operative mortality is much less, although it is still liable to be more than for elective gastrectomy.

Indications for Operation in Gastric and Duodenal Ulcer.

The indications for operation have been discussed extensively by both physicians and surgeons, and the operative procedures have seen much change from the conservative to the radical excision of the stomach. Some indications are absolute, such as pronounced obstruction, large gastric ulcers suggestive of malignancy, penetration with severe bouts of pain, perforation, and repeated severe hæmorrhage especially in the higher age group.

The borderline cases present the problem—the less severe hæmorrhages, the partial response of ulcer pain to medical treatment, the gastric ulcer slightly suggestive of malignant change, the minor degree of pyloric obstruction with imperfect response to medical treatment.

It is not the purpose of this paper to discuss operative technique, but the immediate and remote results are recorded to show that, although most patients derived lasting and almost complete relief of symptoms, a few were left a moderate amount of discomfort. Moreover, the operation was not without risk of life, especially in the older patients. However, with improvement in surgical technique, anæsthesia and resuscitation and the wider range of antibiotics this operative hazard is being reduced. When operation was performed subtotal gastrectomy with gastro-jejunostomy was the operation of choice. Simple gastro-enterostomy was reserved for cases of pyloric obstruction from chronic ulcer occurring in elderly people with low acid secretion.

Twenty-seven patients suffering from duodenal ulcer were subjected to operation, 23 having subtotal gastrectomy, three gastro-jejunostomy and one simple oversewing of the ulcer. In eight of these cases subtotal gastrectomy was decided upon because of a long history culminating in severe persistent pain suggestive of penetration; in ten there was not a clinical story suggesting penetration, but the course was one of repeated relapses and failure of the patient to remain symptom-free out of hospital. Seven patients were operated on because of obstruction, and in three of these cases the operation of gastro-jejunostomy was performed. Thirteen patients from the above-mentioned groups also had a past history of bleeding, and one other patient was operated upon for recurrent bleeding in the absence of dyspepsia.

Of the 23 patients undergoing subtotal gastrectomy, 20 had a good immediate post-operative course; one required urgent laparotomy for a leaking duodenal stump and made a satisfactory recovery; one died from bursting of the duodenal stump, and one died suddenly from aortic stenosis five days after operation.

Eighteen of these patients were studied in our follow-up clinic for an average of two years (range: twelve to thirty-eight months) after operation. Of these 16 were delighted with the result of their operation, enjoying good health and suffering little or no abdominal discomfort—at the most there was occasional fullness after large meals, flatulence or œsophageal regurgitation; one developed an anastomotic ulcer with hæmatemesis and perforation, requiring immediate over-sewing of the perforation and later resection; one complained of frequent bouts of moderately severe dyspepsia, but was found to be consuming very large quantities of alcohol.

Three patients were subjected to gastro-enterostomy in preference to subtotal gastrectomy because they were elderly and had duodenal obstruction with low gastric acidity. They all had a satisfactory immediate post-operative course and their follow-up revealed an excellent result for twenty-six, twenty-one and seven months respectively. They had only minimal epigastric discomfort and gained in weight.

Eighteen patients with gastric ulcer were subjected to subtotal gastrectomy. In six cases operation was performed because there was considerable pain and the ulcer was large, findings suggesting the possibility of malignant change; in three of these cases also the patient gave a history of bleeding. In four cases the ulcer was small, but there was severe and persistent pain indicative of penetration; two of the patients in this group also gave a history of bleeding. In six cases the pain was less severe and not indicative of penetration, but there were repeated

relapses and failure of the patient to remain symptom-free when discharged from hospital; three of the patients in this group also gave a history of bleeding. In two cases operation was performed for pyloric obstruction.

Sixteen patients had a good immediate post-operative course; one developed a perigastric abscess, which discharged externally with satisfactory recovery; one died immediately after operation, probably from ventricular fibrillation.

Sixteen patients were followed up in the out-patient department for an average period of thirty-one months (range: one to five years); one additional patient, a known subject of *tabes dorsalis*, died of pneumonia and renal failure seven months after operation—his dyspepsia had been relieved by operation. Of these 16 patients 14 remained well with either no dyspepsia or only minor discomfort on rare occasions; one had frequent dyspepsia suggesting an anastomotic ulcer, but X-ray examination and gastroscopy revealed no abnormality; one with hypochlorhydria had been subjected to local excision of a small gastric ulcer, but suffered a recurrence at the site of excision—his progress after subtotal gastrectomy was excellent.

Comment.

This study has provided further evidence that, as a group, cases of gastric and duodenal ulcer have many differences, especially with regard to sex incidence and age of onset. It is of interest that while differing in many respects from those of both gastric and duodenal ulcer, the symptoms of chronic gastritis resemble those of gastric ulcer more closely than those of duodenal ulcer. It may be that gastric ulcer and chronic gastritis have features in common in their ætiology.

Although, as a group, both gastric and duodenal ulcer patients conformed to the text-book description, it is of importance that some individual patients gave an atypical history, especially with regard to the food-pain relationship. This variability should be appreciated by the physician when making a diagnosis.

With regard to treatment we realize that a hospital practice favours the reception of ulcer patients with severe symptoms and with complications. Thus a higher proportion fail to respond to medical treatment than would be expected in general practice. However, it is our impression that with improved surgery a greater percentage of patients in the community will benefit from surgical aid. Acute ulcers will certainly heal with simple medical treatment. Large chronic ulcers in the stomach usually require surgical removal. Often small chronic ulcers will heal if given suitable conditions. However, many small chronic ulcers either fail to heal, or healing is only temporary: with each severe relapse the patient becomes more despondent, and perforation or hæmorrhage becomes more likely.

Summary.

1. A group of 93 patients with duodenal ulcer (75 males and 18 females) and 48 patients with gastric ulcer (36 males and 16 females) was studied in order to record the features of gastric and duodenal ulcer as seen in hospital out-patient and in-patient practice in Melbourne, and to serve as a control to a group of patients with chronic gastritis investigated under similar conditions.

2. The average age of onset of symptoms was thirty-six years for duodenal ulcer and forty-four years for gastric ulcer. Most patients came to hospital because of some increase in the severity of the symptoms. A family history of ulcer or dyspepsia was more common in duodenal than in gastric ulcer.

3. Pain was the most common presenting symptom in both groups (83% of the total). It was usually referred to the epigastrium and related to meals, occurring on the average two hours after meals for duodenal ulcer and one and a half hours after meals for gastric ulcer; but in both groups its occurrence varied from immediately after meals to four hours after. It was usually relieved by both food and alkali.

4. Overt hæmorrhage was a common complication of both gastric and duodenal ulcer and was often the reason for the patient's coming to hospital.

5. Fourteen patients with duodenal ulcer had a history of previous perforation. Of these nine failed to respond to medical treatment and required gastrectomy.

6. Histamine test meal examination revealed high levels of free acid and a large volume of secretion in duodenal ulcer, whereas in gastric ulcer the free acid and volume were usually in the normal range, although the results were variable, probably depending on the site and size of the ulcer and the amount of surrounding gastritis.

7. In the treatment; an initial admission to hospital is urged in order to provide every facility for the ulcer to heal. Rest, sedation, alkalis, atropine, iron and an adequate diet are administered. For the subsequent out-patient treatment a plan of observation and treatment is described.

8. The indications and results of surgery are recorded. Subtotal gastrectomy with gastro-jejunostomy has been the operation of choice except for the elderly patient with pyloric obstruction, for whom gastro-jejunostomy alone has been employed. Most patients subjected to surgery experienced considerable and often complete relief of their symptoms, but some immediate and remote complications were encountered.

Acknowledgements.

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SOME RADIOLOGICAL ASPECTS OF TUBERCULOUS DISEASE OF BONES AND JOINTS.[1]

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THE radiological appearances of tuberculous infections involving bones and joints are extraordinarily varied in different cases and at different stages of the disease. In skeletal as in pulmonary disease it is rarely possible, early in the process, to make a positive diagnosis on the evidence presented at a single X-ray examination. However, this first examination will almost certainly serve to eliminate some of the possibilities considered at the initial clinical examination, and will quite often leave little real doubt as to the nature of the lesion.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on September 27, 1951.

The tuberculous process has certain characteristics which influence the radiological picture. The most important is the chronic progressive nature of the disease, and when the process has been watched over a period of time it is this slowly continued development and the relative rate of development of the various features that usually clinch the diagnosis. It is for this reason that progressive radiographic examinations are of such importance in these diseases, and it is impossible to overstress their value. Some indication of the rate of progress to be expected is indicated by the cases discussed.

The initial focus of infection is often situated in the synovial membrane. It is obvious that one cannot hope to demonstrate the first group of tuberculous foci here, as may be possible in the lung, and it is on this account that the radiological latent period in this, a chronic process, may be long. However, the actual demonstration of the organism is also difficult and may not, indeed, be possible at any stage of the disease. It is, then, on the development of radiological signs that we must mostly wait to be able to confirm a tentative clinical diagnosis.

Usually some signs are evident on the radiograph by the time the patient presents for radiological investigation. In this respect it is worth noting that Figures IA, II and IV each represent the first radiograph taken in three cases chosen more or less at random. However, at times these first signs may be very slow in appearing, and rarely they may not be evident for some three months from the commencement of symptoms. In such cases this delay itself, with persisting symptoms, is evidence in favour of tuberculosis. If, then, the examination of a suspected site reveals no abnormality, it should be reexamined three or even four times at intervals of approximately three weeks before the possibility of tuberculosis can be excluded.

The first changes seen on the X-ray film are slight, and may, for as long as several months, be only those due to effusion into a joint or to decalcification. Such changes will be detected earlier by comparison with the opposite joint, and this comparison should always be made where it is applicable.

Lesions of Bone.

Lesions of bone, whether or not they accompany a joint lesion, are in general characterized by a slowly progressive destruction, which will usually continue to progress for at least a month or two despite any treatment.

The major features of the bone lesion may be briefly listed as follows: (i) long course and slow recovery; (ii) little if any sclerosis; (iii) little if any periostitis; (iv) sequestra infrequent and small; (v) penetration of cartilage barriers; (vi) formation of calcifying abscesses. Not all these features will be evident in any one case, but they represent the fundamental guiding principles, the hall-marks of the disease.

When early lesions are detected, the demonstration of a slow evolution over weeks or months is of the utmost importance. At the first X-ray examination some estimate of the chronic nature of the process can be made by correlating the appearances with the duration of the history, and in some circumstances this may be a major consideration in creating a positive diagnosis.

There may, on a single film, be indications as to the chronicity of the lesion. Extensive peripheral osteoporosis or actual bone atrophy (Figure IV) may have resulted from prolonged disuse, or disturbances of growth resulting from local hyperæmia may be evident. It is not unusual to find an epiphysis larger than its opposite number (Figure II), and adjacent ossification centres may appear earlier than expected. Such signs are especially valuable in young infants, in whom the duration of the illness is often a matter for conjecture. Any pronounced soft-tissue wasting will also, of course, be visible on the radiograph.

Destructive areas in bone characteristically extend with little if any marginal sclerosis. This applies almost universally to metaphyseal and epiphyseal lesions. The rare diaphyseal lesion must generally be excepted under this head; but, even in these, non-sclerotic areas often persist over very long periods, and this fact will assist in their differentiation from other sclerotic diaphyseal lesions.

It should be remembered that secondary infection of a lesion may result in the occurrence of much bone sclerosis producing a reversal of the typical picture. It is therefore important for the radiologist to know if sinuses have formed and what surgical procedures may have been undertaken.

In lesions about the epiphyseal lines periosteal reactions are unusual, but they occur sometimes. When present, the calcification resulting is light and irregular in character, and it may show a peculiar slow fluctuation in density from time to time. It is often absent immediately overlying a bone erosion, but will appear close by along the shaft of the infected bone. These reactions never show the rapid increase in density characterizing pyogenic lesions, nor do they ever reach the same degree of density.

Cartilage does little to prevent the spread of the infection, and this fact has its effect on the behaviour of a tuberculous infection. The epiphyseal cartilage in particular offers practically no resistance to the spread of a tuberculous infection. This is in direct contrast to the acute hæmatogenous type of osteomyelitis and is often a feature of the X-ray picture. However, a subacute type of pyogenic infection arising in the metaphysis will also penetrate the epiphyseal plate sometimes, and such lesions, at least in their early stages, may be quite indistinguishable from a similarly situated tuberculous lesion, excepting only that the surrounding osteoporosis is likely to be less pronounced. In spinal cases the intervertebral disk is destroyed early, so that narrowing of the disk space and destruction of adjacent edges of two vertebral bodies are often the outstanding features of the early radiograph. Destruction of the disk with narrowing may be the first sign, occasionally preceding demonstrable bone destruction by several weeks. The problem as it relates to articular cartilage will be mentioned later.

Tuberculous infections of bone differ considerably from the pyogenic infections in the reluctance of the former to produce sequestra. When these do form, it is within a large area of bone destruction (Figure VI), and they therefore arise late. They are never very large. They do not proceed to produce the dense angular shadows seen so often in pyogenic infections, but are of such poor density that they may be rather difficult to distinguish. Their borders are serrated and hazy.

The most characteristic single feature of the disease is the tendency to form large calcifying abscesses (Figure V). These abscesses are not, of course, calcified when first formed, but are notable for their long duration and often for their unexpected size, in view of the extent of the bone lesions present. They characteristically spread downwards in the tissue planes, show a rounded lower border which may or may not be visible radiographically, and may, of course, give rise to sinuses. Those which end blindly within the soft tissues will sooner or later show speckled calcification at the lower end, or they may show a slow homogeneous increase in density. Both types tend eventually to produce a dense, heavily mottled shadow with a clear-cut border, the appearance of the whole structure being entirely characteristic.

Lesions of Joints.

As far as the radiological evidence is concerned, it is mostly impossible to decide whether a lesion involving a joint originated in that joint or in adjacent bone. However, when the involvement of the joint is the outstanding feature of the disease, the sequence of events tends to follow a certain pattern, the salient points of which are as follows: (i) Continuous development of (*a*) effusion into joint, (*b*) decalcification. (ii) After some months, (*a*) destruction of adjacent bone, (*b*) thinning of joint space. (iii) After some years, ankylosis.

The presence of an effusion is indicated by the faint but sharply defined soft-tissue shadow cast by the distended joint capsule and is usually accompanied by some slight

widening of the joint space (Figures I and IV). Its recognition is usually easy in the more superficial joints, but may be difficult in joints such as the hip joint. The occurrence of this effusion often constitutes the first visible sign. It almost invariably accompanies the early lesion and will persist for months. It is accompanied before very long or from the beginning by decalcification of bone. This feature is well demonstrated in the cases discussed. The decalcification may be described as being of two types. Local decalcification of bone in the immediate vicinity of the joint is usually seen first. This is presumably a result of continued local hyperæmia, and will be more pronounced than could be accounted for by disuse alone. It is a very important feature of the early lesion. In the carpus and tarsus it may become so intense that the outlines of the various bones can hardly be discerned. The second type of decalcification developing more or less concurrently is the osteoporosis of disuse which extends distally.

Bone destruction occurs later, sometimes much later, and will show the features already discussed. More often than not, some bone destruction will be evident by the time of the first radiological examination. This is due partly, no doubt, to the insidious onset of symptoms, and partly to the fact that a proportion of the lesions originates in bone. Which is the chief factor is difficult to determine.

The extent to which bone destruction may ultimately proceed in the hip joint is well shown in Figure III. This destruction will occasionally be separated from the affected joint by apparently healthy bone (Figure IA).

It is obvious from what has been said that the articular cartilage does not form a very effective barrier to the spread of the tubercle bacillus from joint to bone or *vice versa*. Despite this, extensive destruction of the articular cartilage with narrowing of the joint space is late in appearing, but it may precede extensive bone destruction and be an outstanding feature of the picture for a considerable time (Figure IV). In pyogenic arthritis, destruction of articular cartilage will be much more rapid in terms of time; involvement of bone, pronounced soft-tissue swelling *et cetera*, also appear much more rapidly, so that the thinning of the joint space is not a prominent feature.

Healing.

When a joint is involved, healing is accompanied in the vast majority of cases by ankylosis, fibrous or bony. Bony ankylosis is demonstrated radiographically by the continuity of bone trabeculæ across the joint. This is usually recognized readily, but sometimes several views will be necessary to ensure that a false appearance of ankylosis is not produced by overlapping bone shadows. Apart from the demonstration of ankylosis, healing is evidenced by return of the calcium content of the parts to normal and by the reestablishment of a clear and sharply defined cortical and trabecular pattern. An irregular widening of the trabecular network remains, usually for life.

Sometimes, despite all other evidence indicating quiescence, rarefied areas will persist. In these cases an absence of any change over at least two years should be demonstrated before the lesion is pronounced healed.

Conclusion.

No attempt has been made to deal with all aspects of this subject. The object has been to give some impression of the subject as it confronts the radiologist and to indicate something of the principles on which his reasoning is based.

It should be stressed that each radiograph represents a single phase in the evolution or regression of the lesion. Therefore, in order to obtain from it the greatest amount of information, it must be studied in conjunction with the clinical signs and symptoms or in its proper context with a series of radiographs taken over a period of time.

In those cases in which the diagnosis remains in doubt, it is the consideration of the time factor that is crucial.

Legends to Illustrations.

FIGURE IA.—Initial radiograph of child, aged twenty months: slight widening of the joint space, slight local decalcification and early evidence of a metaphyseal bone lesion.

FIGURE IB.—Same patient as in Figure IA, ten months later: extension of bone destruction, still evidence of effusion, local decalcification more pronounced.

FIGURE II.—Initial radiograph of child, aged six years, with a history indicating a duration of four months; local decalcification, bone erosion in acetabulum and femoral neck, some narrowing of the joint space. The epiphyses for the head and greater trochanter are each a little larger than on the opposite side.

FIGURE III.—Same patient as in Figure II, after six years' treatment; very extensive bone destruction and atrophy of the femur.

FIGURE IV.—Initial radiograph of child, aged five years; local and peripheral deossification, bone destruction, narrowed joint space and bone atrophy; duration unknown, but probably some months. The outline of the distended joint capsule is evident.

FIGURE V.—Same patient as in Figure IV after two years' treatment. A calcified abscess extends into the pelvis, and also visible is the upper end of another which reached halfway down the thigh.

FIGURE VI.—Child, aged ten years, under treatment for four years. A saddle-shaped sequestrum is present within the large eroded area involving the roof of the acetabulum.

Reports of Cases.

SPONTANEOUS RUPTURE OF THE APPARENTLY NORMAL SPLEEN: REPORT OF A CASE OF ITS OCCURRENCE IN A PATIENT SUFFERING FROM A GENERALIZED ILLNESS OF DOUBTFUL ÆTIOLOGY.

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COMPARED with traumatic rupture of a normal or diseased spleen, spontaneous rupture of the spleen is exceedingly rare, especially in the case of the normal spleen.

Cases of spontaneous rupture of the spleen may be grouped as follows (see Table I):

1. When the spleen is diseased: (*a*) in the case of generalized illness with involvement of the spleen; (*b*) in the case of local disease of the spleen only.

2. When the spleen is normal: (*a*) during the performance of a physiological act; (*b*) with no apparent cause at all.

3. When the spleen is apparently normal in a patient with a generalized illness, as in the case here reported.

Spontaneous rupture of the diseased spleen and of the normal spleen during the performance of a physiological act is discussed sufficiently by the authors of the articles listed in Table I.

Spontaneous rupture of the normal spleen with no apparent cause is very rare indeed; in fact, Lundill (1934) said that a normal spleen would not rupture spontaneously, and Susman (1927) doubted its probability. However, Chamberlain (1950) described the following case:

Without any previous illness or trauma, his patient collapsed at work and was diagnosed as suffering from a perforated peptic ulcer. The spleen was found to be normal on macroscopical and microscopical examination, except for the presence of small tears in the capsule, from which hæmorrhage was occurring. The patient died a few hours after operation, and even an autopsy did not establish any disease of the spleen or other organ.

Zuckerman and Jacobi (1937) collected 28 cases of spontaneous rupture of a normal spleen from the literature, but regarded only 21 as genuine. There are numerous other reports of single cases, as, for instance, those of McCoy (1911), Bailey (1930), Dahle (1934), Watson and Ferdeber (1942), Jones (1944) and Silverman and Randazzo (1946). Consequently one must now agree with Ellison (1949) that this condition is a clinical entity. The mechanism of the rupture of a normal spleen without obvious cause is undetermined. Aird (1949) suggests that there may be congestion which disappears before operation. This is, of course, only an hypothesis.

Clinical Record.

This is a case of spontaneous rupture of an apparently normal spleen occurring during the course of an illness characterized by malaise and raised temperature, wound dehiscence, chronic lung and blood changes and periarthritis of an ankle joint, with recovery of the patient.

TABLE I.
Spontaneous Rupture of the Spleen.

State of the Spleen.	Other Factors.
Diseased spleen.	(*a*) Generalized disease with involvement of the spleen: Malaria (Noland and Watson, 1913; Russ and Gaynor, 1945; Lubitz, 1949; Johnson, 1950). Infectious mononucleosis (McCarthy and Knoepp, 1944; Darley *et alii*, 1944; Spence, 1947). Typhoid fever (Connor and Downes, 1914). Leuchæmia (Rubnitz, 1943). Sarcoidosis (James and Wilson, 1946). Arterial disease (Black, 1933; Coleman, 1939). Hæmophilia (Susman, 1927). (*b*) Local disease of the spleen: Sarcoma of the spleen (Littlefield, 1946). Banti's disease (Lubitz, 1949). Torsion (Lubitz, 1949). Degeneration, perforation, abscess, hydatid disease (Susman, 1927).
Normal spleen.	(*a*) During the performance of a physiological act: Bending to lift a heavy weight (Susman, 1927). Coitus (Stretton, 1926). Normal labour (Schwing, 1880). Defæcation (Druitt, 1947). (*b*) With no apparent cause at all: In pregnancy (Hubbard, 1879). Whilst resting (Bailey, 1930; Jones, 1944). At ordinary work (Chamberlain, 1950). Whilst sleeping (McCoy, 1911; Watson and Ferdeber, 1942).
Apparently normal spleen.	Generalized disease, as in the case here reported.

The patient was a man, aged fifty-eight years, who was admitted to the Royal North Shore Hospital of Sydney on August 31, 1946, complaining of severe abdominal pain of eight hours' duration.

His history was that he had been well until three days prior to admission to hospital, when he commenced to suffer from an illness characterized by headaches and generalized aches and pains. On the day of admission, whilst he was in bed, these pains gradually increased in intensity and became localized in the upper part of the abdomen, more especially in the left hypochondrium. Eight hours later they became so severe that he collapsed and was admitted to hospital with the diagnosis of ruptured peptic ulcer. There were no other relevant symptoms save that he had been constipated for the previous two days.

Investigation of his past history revealed that he had always been a thin cadaverous man, accustomed to flatulence and feelings of abdominal distension and constipation. Eight years previously he had had symptoms of epigastric pain relieved by food. A barium meal examination revealed no evidence of peptic ulcer, but a fractional test meal examination was said to show hyperacidity. This illness lasted for six months, the patient being treated with dietetic measures and alkaline mixtures. The pains recurred at irregular intervals for five years. The pains were different from those from which he now suffered.

One month prior to admission to hospital he had had an infected abrasion of the right hand after gardening. There was no involvement of the draining lymph nodes. Though he was treated with sulphadiazine, the abrasion took three weeks to heal.

There had never been any trauma likely to cause rupture of the spleen.

Examination of the patient on admission to hospital disclosed a shocked, pale, emaciated man. His tongue was dry and furred. His pulse rate was 130 per minute, his respiratory rate 22 per minute and his temperature 97° F. His blood pressure was 75 millimetres of mercury, systolic, and 50 millimetres, diastolic.

The abdomen was a little distended and there was no movement on respiration. There was generalized abdominal tenderness and rigidity, greatest in the upper part of the abdomen. Percussion revealed shifting dulness in both flanks, and auscultation revealed that there were no bowel sounds. A rectal examination revealed no abnormality.

There were no other relevant findings. The scratch on the dorsum of the right hand had healed without any scarring. Urine examination revealed no abnormal constituents.

The clinical picture was one of blood loss rather than of a perforated hollow viscus. The shocked state was treated by blood transfusion and the stomach kept empty by means of a Ryle's tube.

Three hours after his admission to hospital, when his condition had improved, laparotomy was performed under general anæsthesia ("Kelene" and intratracheally administered ether). The abdomen was entered through an upper right paramedian incision. This revealed much free blood and clot in the peritoneal cavity. There was an almost complete tear through the diaphragmatic surface of a normal-sized, normal-looking spleen down to its hilum. All other organs were normal.

Splenectomy was performed and a further transfusion of blood was given.

Early convalescence was aided by blood transfusions and an indwelling Ryle's tube. On the sixth post-operative day wound dehiscence occurred, though there had been no post-operative vomiting or distension. There was a complete failure of all layers of the wound to heal and no gross evidence of infection. The wound was resutured with interrupted sutures of fine tantalum for the peritoneum and stainless steel for the fascia. It then healed normally without any hernia formation.

Recovery was slow, the patient suffering from extreme weakness and lethargy with a constant feeling of malaise. In the immediate post-operative period he had a persistent temperature rise up to 102° F. each evening with a fall to 99° to 100° F. each morning. This temperature rise (which was unaffected by his wound dehiscence) lasted until December, 1946, when it declined by lysis. No cause could at first be found for this raised temperature. Blood counts were at first normal, except for the usual post-splenectomy leucocytosis (see Table II). Repeated blood culture, a Paul-Bunnell test and agglutination tests for typhoid, the paratyphoids and brucella organisms yielded negative findings. The results of a Wassermann test and a complement deviation test for gonorrhœa were negative.

On October 7, 1946, five weeks after operation, chest signs first developed. There was a persistent cough with a small amount of white sputum, which by some oversight was never submitted to culture. Dulness and crepitations appeared at the left lung base posteriorly. Fluid collected there in the pleural cavity, and on October 17, 600 millilitres of straw-coloured sterile fluid, which contained a few pus cells only, were removed. The fluid did not collect again, and X-ray examination then showed a haziness over the left lower lung field. The diaphragm was not raised and there was no evidence of subphrenic abscess.

Over the next few weeks this infiltration involving the left lung gradually cleared up completely. As it resolved, however, a similar process occurred in the right lower lung field, without any pleural reaction this time. Each film showed, in addition, some old scarring at both apices. The right lung gradually cleared, so that by January, 1947, four and a half months after operation, chest X-ray examination showed only some residual pleural thickening in the left costo-phrenic angle and some residual pneumonic changes at the right lung base (which, incidentally, were replaced four months later by fibrosis and some small bullæ). As the lung lesions subsided, so did the temperature fall and the patient's general condition improve.

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TABLE II.
Blood Examination Findings.[1]

Date.	Hæmoglobin Percentage.	Erythrocyte Count (per Cubic Millimetre).	Total Leucocyte Count (per Cubic Millimetre).	Absolute Number of Neutrophile Cells (per Cubic Millimetre).[2]	Differential Leucocyte Count (Percentages).									Remarks.
					Neutrophile Cells.	Eosinophile Cells.	Basophile Cells.	Lymphocytes.	Monocytes.	Band Forms.	Metamyelocytes.	Smudge Forms.	Unclassifiable.	
4/ 9/46	90		13,900	11,467	82·5	1·0		6·0	7·5	2·0	1·0			
5/ 9/46	92		13,500	11,340	84·0			9·0	5·0	1·0	1·0			
13/ 9/46			11,600	9,280	80·0	2·0		14·0	3·0	1·0				
8/10/46			8,800	7,040	79·5			17·5	3·0					
11/10/46			13,000	7,860	62·0	1·0		30·0	7·0					
16/10/46	81	$4·1 \times 10^6$	25,000	7,800	32·5			17·5	4·0	3·5		32·0	10·5	
21/10/46			19,900	6,800	34·5	0·5		34·5	4·0	2·5		17·5	6·5	
24/10/46			29,800	5,662	19·5			24·0	2·0	3·0		28·0	23·5	The unclassified cells are very immature cells of the non-granular series.
29/10/46			20,900	4,589	21·5	2·0		38·5	1·0	2·0		30·0	5·0	
1/11/46	73	$3·5 \times 10^6$	24,300	10,692	44·5			26·5	2·0	7·0		15·0	5·0	
5/11/46			14,100	4,900	35·5	0·5		43·5	4·0	1·0		14·5	1·0	
11/11/46			10,500	1,575	15·0	0·5		49·0	3·0	1·0		27·5	4·0	
12/11/46	65		13,000	4,420	34·0	0·5	0·5	44·0	2·0	2·0		16·0	1·0	
19/11/46			10,000	3,200	32·0	2·0		47·0	3·0	4·0		12·0	—	
29/11/46	67		12,000	5,760	48·0			49·0	3·0					
17/12/46			9,100	2,820	31·0	2·0		55·0	12·0					
3/ 1/47	82		10,800	4,300	43·0	3·0	1·0	46·0	7·0					
14/ 5/47	99	$4·86 \times 10^6$	8,000	2,720	34·0		0·5	50·0	8·0				7·5	Unclassifiable and immature mononuclear cells, some of which have nucleoli. They are not typical lymphoblasts.
1/ 8/47	96	$5·0 \times 10^6$	13,000	5,330	41·0	1·5		43·5	14·0					
27/10/47	101		11,900	5,280	44·0	2·5	1·0	40·0	12·5					
6/ 3/48			6,900	1,552	22·5	2·0		64·0	11·5					
24/ 4/48			7,100	1,518	37·0	0·5	0·5	49·0	13·0					
25/ 6/48			5,500	2,585	47·0	1·0		44·0	8·0					
12/12/51	98	$5·0 \times 10^6$	7,350	4,183	58·0	2·0		25·0	14·0	1·0				

[1] Blood platelets and bleeding and coagulation times were always normal.
[2] Normal neutrophile cell count 3,000 to 5,800 per cubic millimetre.

During November, 1946, convalescence was also complicated by a painful periarthritis of the left ankle joint, which slowly subsided in a month. X-ray examination of the ankle showed that the bones were not affected. The blood uric acid content was not raised.

Treatment with penicillin and sulphadiazine had no effect on the course of the chest or ankle lesions.

During this time the peripheral blood count showed interesting changes (see Table II). Anæmia of low colour index was present for four months after operation and was treated with blood transfusions and iron. It responded only very slowly to treatment, but since January, 1947, the red cell count has always been normal. The white cell count showed interesting changes, which started with the onset of lung involvement. There was first a neutrophile leucocytosis in the first month after the splenectomy. Then, as the chest signs appeared, the total white cell count increased and many smudge and unclassifiable forms appeared in the peripheral circulation. They appeared to be large immature cells of the non-granular series. Though the relative percentage of neutrophile cells dropped, the absolute number remained mostly higher than normal. (Unfortunately, the patient persistently refused sternal puncture or marrow biopsy.) A recent blood film (December, 1951) showed a normal leucocyte count.

The patient was discharged to the out-patient department on January 19, 1947, four and a half months after operation, feeling much improved.

The patient has been followed up at regular intervals to the present, last being examined in January, 1952, at the age of sixty-three years. He was still well, though of the same emaciated appearance. He stated that he tired easily and suffered from palpitation of the heart. However, clinically the cardio-vascular system was normal and there was no hypertension. He still had attacks of abdominal distension and constipation. He had developed bilateral inguinal herniæ, which did not incommode him. Otherwise the findings of a general examination were normal. An X-ray examination of the chest showed some bullæ and fibrosis at the right lung base with the same scarring at both apices. A blood count showed a normal red cell count and hæmoglobin value, but the white cell count was within normal limits. There were no unclassifiable white cell forms. The wound was well healed, with no evidence of hernia formation. However, he had to have several stainless steel sutures removed owing to the pain felt from their presence.

Dr. A. J. Canny (now Professor of Pathology at the University of Queensland) examined the spleen and stated that there was no abnormality in the splenic structure adequate to account for the rupture.

Comment.

Though the spleen was normal both macroscopically and microscopically, there may have been some change in its tissues not discernible by ordinary methods of investigation, so that the normal may have been only apparently normal. It has been noticed, too, that in many of the reported cases of spontaneous rupture of the normal spleen the catastrophe is preceded for some days by epigastric pains of varying intensity as in the case here discussed.

The question in this case is the relationship between the general illness and the rupture of the spleen. The splenic rupture and the chest and blood changes were separated by an interval of five weeks, but this was bridged by the patient's general ill health and the raised irregular temperature. Almost certainly the wound dehiscence must be included as a part of the general picture, as the wound showed a complete failure to heal without obvious cause, such as gross infection or increased intraabdominal tension.

The blood changes, of course, could be explained as normal post-splenectomy changes having nothing to do with his illness. Singer, Miller and Dameshek (1941) found that splenectomy was usually followed by leucocytosis with a relative and occasionally an absolute reduction in the number of neutrophile cells and an increase in lymphocytes and monocytes. Wintrobe (1946) states that abnormalities of the blood may persist for many years after the removal

of a normal spleen in man, as in this case. Jordan and Robeson (1942) reported the production of lymphoid nodules in the bone marrow of domestic pigeons following splenectomy. Should a similar condition occur in man, it would help explain the blood changes. Possibly, too, lymphoid infiltration of other organs, including the lungs, could occur. However, this would not explain the temperature and general malaise suffered by the patient whose condition seemed to be of infective, probably viral, origin. At no time did the clinical picture ever resemble infectious mononucleosis, which explains why the Paul Bunnell test was not repeated.

Thus, neither the cause of the rupture of the spleen nor the general illness of this patient was able to be determined, nor was their relationship to each other.

Summary.

Spontaneous rupture of the spleen is briefly discussed.

A case is reported of a patient whose illness commenced with general malaise followed by spontaneous rupture of an apparently normal spleen. Convalescence from the splenectomy was prolonged, and the patient was four and a half months in hospital owing to general malaise, wound dehiscence, involvement of both lungs, periarthritis of an ankle and changes in the peripheral blood count, including anæmia and leucocytosis, with an increase in the number of the cells of the non-granular series, many of which were immature.

The cause of this illness, as well as the reason for these changes and their relationship to the rupture of the spleen, was not able to be determined.

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I wish to thank Dr. E. Beatrix Durie, bacteriologist to the Institute of Medical Research, for her interest in the investigation of this patient and her advice in the preparation of this paper. I wish also to thank Miss D. Drake, of the Unit of Clinical Investigation, for her clerical help in the preparation of this paper, and Miss A. Scougall for her help with the literature.

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Reviews.

VITAMINS.

FOR many years Dr. Leslie J. Harris has been outstanding as a research worker and writer on vitamins. In his latest book, "Vitamins: A Digest of Current Knowledge", he amply maintains his position.[1] This is a small book of 244 pages, but it is amazing the amount of accurate and up-to-date information on the subject of the vitamins that has been included. There is brief but, for most readers, ample information relating to the sources, properties, synthesis, formulæ and medical uses of all the recognized vitamins. A short history of the discovery of the vitamins is followed by separate sections on each of the vitamins. The generally recognized symptoms and signs of deficiencies of each are given, but it is noted that care has to be taken in interpretation, for some of the signs may not be specific. It is not, however, sufficiently stressed that deficiencies of members of the *B* group are probably always multiple. The book is illustrated with well-selected pictures of the effects of deficiencies. While the information given is necessarily short, it is as nearly up to date as possible, and there is a very useful bibliography, so that one can readily follow up any particular interest. The book can be recommended to all interested in the vitamins.

MODERN DIETARY TREATMENT.

SIGNIFICANT developments in the science of nutrition and particularly their application to dietary therapy have occurred in the ten years since the previous edition to warrant the publication of a third edition of "Modern Dietary Treatment" by Abrahams and Widdowson.[2]

This new edition presents the material in five areas of the subject. The first three chapters briefly cover the principles of nutrition. In the description of deficiency diseases the authors have made the error, occasionally still seen, of giving the classical descriptions of the frank forms of the diseases, conditions that are rare in England, Australia and similar countries, and have omitted descriptions

[1] "Vitamins: A Digest of Current Knowledge", by Leslie J. Harris, Sc.D., D.Sc., Ph.D., F.R.I.C.; 1951. London: J. and A. Churchill, Limited. 7½" × 5", pp. 256, with 84 illustrations and 111 structural formulæ. Price: 15s.

[2] "Modern Dietary Treatment", by Margery Abrahams, M.A. (Oxon.), M.Sc. (Columbia University), and Elsie M. Widdowson, D.Sc., Ph.D. (London); Third Edition; 1951. London: Baillière, Tindall and Cox. 8" × 5½", pp. 362. Price: 21s.

of the minor forms, which are more likely to be seen by the majority of the readers. This is the weakest section of the book.

Some sixty pages are devoted to straightforward explanations of the types of diets recommended for various diseases. Then follows the major portion of the book, containing detailed descriptions of diets for use in all the diseases requiring special diets. Although prepared primarily for use in England, most of the diets could be used without modification in Australia. There is a short section on recipes which are given in such general terms as also to be of use in Australia. The final section contains a number of tables, including the chemical composition of foods, information about vitamins, recommended dietary allowances, Calorie requirements in relation to age and sex.

This edition should prove as useful as the other editions to doctors and nurses, especially to those who have to advise and devise diets for their patients.

"CHIROPODIAL ORTHOPÆDICS."

THE novel title of "Chiropodial Orthopædics" is given to a recent book by Franklin Charlesworth.[1] The subject covers the manufacture and fitting of various protective appliances that are used to relieve painful conditions of the feet. In most cases this entails the making of a cast of the foot, and the author has described in detail his methods. Insoles and supports are illustrated to fit and to relieve almost every pathological condition of the foot: not only the common disabilities but also gross deformities both congenital and acquired, where surgery is not indicated or has failed. The author is a specialist in designing footwear and he describes his craft clearly and thoroughly. The book will be invaluable to chiropodists who intend to practise this part of their trade. No one can deny the importance of securing comfort for those with painful feet, and this book makes a definite contribution to the solution of some of the problems that an orthopædic surgeon has to face.

Notes on Books, Current Journals and New Appliances.

MEDICAL LIBRARIES IN HOSPITALS IN GREAT BRITAIN.

A BOOK and periodical list has been compiled by a subcommittee of the medical section of the Library Association in Great Britain.[2] The object is to provide a select list of books in the English language which are in steady demand, giving alternative titles under each of the main divisions of the specialties of medicine. It is pointed out that the number of titles must be strictly limited; at the same time, it is stated that while the inclusion of any book necessarily signifies approval, omission does not imply the contrary. The list is for use in hospital libraries, and it is assumed that publications not mentioned in the list will be available in a large central library. As a matter of fact one wonders why this list has been submitted for review. The books in the list are grouped under the headings of special subjects. English books are chosen for preference, and American works are marked with an asterisk. No such distinction is made in regard to periodicals. These are grouped under various headings, and the list does not particularly arouse our enthusiasm. Not one journal from the Southern Hemisphere is included in it. Hospital workers who wish to read a communication from "down under" will have to visit central libraries, which doubtless are sufficiently numerous. This is a challenge to workers in the Southern Hemisphere to increase the value of their own journals by publishing their work in their own countries. From this list prepared for British hospitals some prominent American journals are missing. The committee has also avoided invidious distinctions by omitting from the list the names of all journals produced by British hospitals.

[1] "Chiropodial Orthopædics", by Franklin Charlesworth, F.Ch.S., with a foreword by Harry Platt, M.S., F.R.C.S., F.A.C.S.; 1951. Edinburgh: E. and S. Livingstone, Limited. 9" × 6", pp. 264, with 131 illustrations. Price: 25s.

[2] "Books and Periodicals for Medical Libraries in Hospitals", compiled by a Subcommittee of the Medical Section of the Library Association; 1952. London: The Library Association, Chaucer House, Mallett Place. 8¼" × 5½", pp. 16. Price: 2s. net.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Text-Book of Ophthalmology", by Stewart Duke-Elder, K.C.V.O., M.A., LL.D., D.Sc. (St. And.), Ph.D. (London), M.D., F.R.C.S., Hon. D.Sc. (Northwestern), D.M. (Utrecht), F.R.C.S. (Edinburgh), F.A.C.S.; Volume V: The Ocular Adnexa; 1952. London: Henry Kimpton. 10" × 7", pp. 1114, with 1181 illustrations, including 32 in colour. Price: 90s.

This volume deals with the ocular adnexa and has chapters on developmental anomalies, and with diseases of the lids, the lachrymal apparatus, the orbit and the paraorbital regions.

"Liver Injury: Transactions of the Tenth Conference, May 21-22, 1951, New York"; 1951. New York: Josiah Macy Junior Foundation. 9" × 6½", pp. 320, with 42 illustrations. Price: $3.75.

The subjects discussed include serum proteins and lipids, the action of lipotropic factors in animals and man, experimental ischæmia of the liver and hepatic coma, the microanatomy of the hepatic vascular system and the effect of insulin on the liver in normal and diabetic man.

"The British Pharmaceutical Codex, 1949. Supplement 1952", published by direction of the Council of the Pharmaceutical Society of Great Britain; 1952. London: The Pharmaceutical Press. 9" × 5½", pp. 160. Price: 25s. net.

In the compilation of this work the Council of the Pharmaceutical Society has relied on the Codex Revision Committee which has had the assistance of eight subcommittees.

"Osler: The Man and the Legend", by W. R. Bett; 1951. London: William Heinemann (Medical Books), Limited. 7½" × 5", pp. 132, with six illustrations. Price: 15s.

The author has tried to portray Osler "in some dozen aspects which are common to him and to those who knew him not in person".

"The Asthmatic Child: The Prevention of Asthma by Simple Home Methods", by G. F. Walker, M.D., F.R.F.P.S.G., D.C.H., M.R.C.P.; Second Edition; 1952. Bristol: John Wright and Sons, Limited; London: Simpkin, Marshall, Limited. 8½" × 5½", pp. 20, with 24 illustrations. Price: 2s. 6d.

The first edition appeared in 1950.

"A Color Atlas of Morphologic Hematology, with a Guide to Clinical Interpretation", by G. A. Daland, B.S., edited by T. H. Ham, M.D.; 1951. Massachusetts: Harvard University Press (Geoffrey Cumberlege); Melbourne: Oxford University Press. 10¾" × 8¾", pp. 74, with nine figures and fourteen coloured plates by Etta Piotti. Price (Australian): 52s. 6d.

The purpose of the book is "to furnish a guide for reference in the study of films of the peripheral blood stained with Wright's stain".

"The Thoracic Surgical Patient: Preoperative, Anesthetic and Postoperative Care", by L. A. Hockberg, M.D., with a foreword by F. B. Berry, M.D.; 1952. New York: Grune and Stratton. 9" × 6", pp. 376. Price: $8.75.

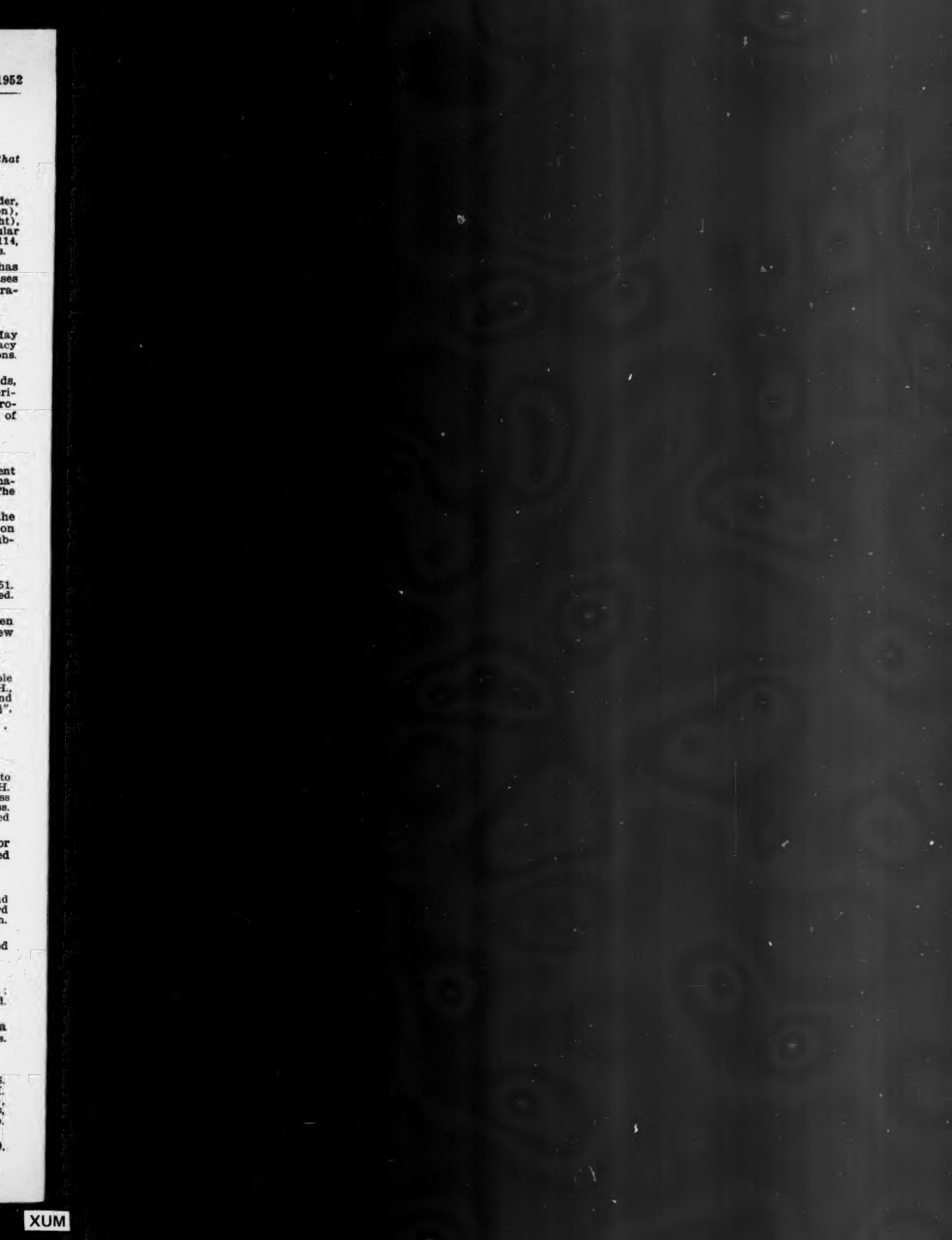
The book is "not intended specifically for the accomplished thoracic surgeon".

"Fluid Balance: A Clinical Manual", by C. A. Moyer, M.D.; 1952. Chicago: The Year Book Publishers, Incorporated. 7" × 4¾", pp. 191. Price: $3.75.

The author's aim is to present a simple practical schema of diagnosis pertaining to fluid and electrolyte imbalances.

"Emergency Surgery", Part IV, by Hamilton Bailey, F.R.C.S. (England), F.A.C.S., F.I.C.S., F.R.S.E., assisted by N. M. Matheson, M.B., Ch.B., M.R.C.P. (London), F.R.C.S. (England), F.A.C.S.; Sixth Edition; 1952. Bristol: John Wright and Sons, Limited; London: Simpkin, Marshall, Limited. 10" × 6½", pp. 197, with 311 illustrations, some in colour. Price: 21s.

Part III of the sixth edition was published in March, 1950.



The Medical Journal of Australia

SATURDAY, MAY 3, 1952.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

A NEWLY ADOPTED METHOD OF ARTIFICIAL RESPIRATION.

It has been recently announced in the daily Press that the National Council of the Surf Life Saving Association of Australia has decided to adopt a back pressure-arm lift method of manual artificial respiration (described as the Holger-Nielsen method) for a trial period of twelve months. This follows the endorsement of the method by the Council on Physical Medicine and Rehabilitation of the American Medical Association, the American National Red Cross and many other bodies in the United States of America, including the Armed Forces, the Federal Civil Defense Administration and the Public Health Service. This endorsement, in which the method is described as being "for use by the general public when dealing with cases of asphyxia", in turn followed a recommendation of the method released on December 3, 1951, by the American National Red Cross, the National Research Council, the Department of Defense, and the Public Health Service in the United States. The adoption of the method is significant in at least two ways. First, it recalls the melancholy fact that a variety of bodies, and notably those concerned with civil defence, are seeking simple and efficient methods of resuscitation suitable for general use in the event of major disasters. Second, it marks the replacement of the Schafer method, which has had a long vogue. The Chief Superintendent of the Surf Life Saving Association of Australia has been quoted[1] as saying that the new method was adopted "without prejudice to the Schafer method". Nevertheless, in the words of a joint statement[2] issued by the Council on Physical Medicine and Rehabilitation of the American Medical Association and the American National Red Cross, "during recent years a growing mass of evidence has indicated that several methods of approximately equal merit in meeting physiological needs are superior to the prone pressure [Schafer] method, and that among them the back pressure-arm lift method best meets the requirements for general public use". Important work on the assessment of the various methods of artificial respiration was referred to in these columns on May 12, 1951. In the reports then quoted, it was stated that the method involving hip lifting and prone pressure was generally most effective; the modification of this in which the "rolling" method of lifting one hip is substituted for lifting of both hips was described as easier for sustained operation, with much to commend it; the Nielsen (arm lift-scapular pressure) method was stated to produce comparable amounts of ventilation, but to be not free from possible risk of shoulder injury. However, when one of the essential criteria is ease of application by the general public, the back pressure-arm lift method appears the most satisfactory. The technique of the method ("essentially the Holger-Nielsen method") is given in the recent American joint statement:

> The subject is placed prone, elbows bent, arms overhead with one hand upon the other. The cheek is placed on the hand, the face turned slightly to one side. The operator kneels on one knee at the head of the subject and puts the foot of the opposite leg near the elbow. He places his hands on the subject's back, in such a way that the thumbs just touch, the heels of the hands being just below a line running between the armpits. He rocks slowly forward, elbows straight, until his arms are aproximately vertical, exerting steady pressure upon the chest. Then he commences to rock backward slowly and slides his hands to the subject's arms just above the elbows. He raises the arms until resistance and tension are felt at the subject's shoulders. Then he drops the arms. This completes a full cycle. The cycles are repeated 12 times per minute, the expansion and compression phases being of equal length, the release periods being of minimum duration.

It is pointed out in the joint statement that the method probably is not quite as easy to carry out and to teach as the prone pressure (Schafer) method, but yet is easy. A child or a frail person can administer the method to a heavy person. It has been taught and used in Europe, especially in Denmark and Norway, for many years. In answer to specific inquiry, the Red Cross Societies of these nations state that there have been no injuries during administration of the method, either in teaching sessions or in actual resuscitation efforts. Studies carried out in the United States generally confirm these findings. Thus adoption of the method for general public use is recommended in the joint statement on the basis of the scientific evidence that it "satisfies the physiological requirements essentially as well as any other studied and has advantages over the prone pressure method, and . . . is a feasible one for use under field conditions of asphyxia and for a broad educational program". It will be interesting to learn the opinions formed after twelve months' trial of the method by such an experienced body as the Surf Life Saving Association of Australia.

[1] *The Sydney Morning Herald*, March 17, 1952.

[2] *The Journal of the American Medical Association*, December 8, 1951.

PATENT MEDICINES AND AUTODIAGNOSIS.

The boundary between necessities and luxuries is a shifting one and hard to fix. As culture advances more and more of what were formerly regarded as appurtenances of wealth and privilege enter the lists of the necessary. Physiological requirements do not dictate form and adornment of homes, colour and shape of clothes nor even the selection of human food. No bodily advantage can be ascribed to the use of tea and tobacco, but a government which attempted to prohibit their importation would have

a short life despite the great easing of the financial position which would assuredly ensue; what Matthew Arnold said of wine applies to tea and tobacco—they add to life's agreeableness, and what adds to life's agreeableness adds to its resources and its power. Some items of luxury belong to the realm of the æsthetic; a community or stratum of population without music is a community or stratum without civilization. Some are to be classified as means of escape from a monotonous or fatiguing environment. A Manchester cynic declared that he was addicted to alcohol because it was the easiest and cheapest way out of Manchester. When we come to patent medicines and most popular drugs and appliances, we find no real pretext or apology for their use; their purchase is from the economic standpoint sheer waste, and yet a considerable fraction of the wage is spent on them. Throwing away good money is not by any means the worst feature of this evil custom; it is true that a bottle of patent medicine may really have a value in pence equal to the shillings demanded by the retailer, but here the heavy costs of advertising and wrapping have to be taken into consideration. What the medical man deplores is the autodiagnosis and consequent autotherapy.

Diagnosis is the most difficult acquirement in medical training and experience, but the layman who treats himself simply accepts the signs and symptoms from an advertisement and proceeds to purchase and consume a drug or mixture without any expert examination being attempted. He, for example, may become aware of undue dyspnœa on exertion; it is to him just something wrong with his bellows, and after a search through advertising literature he selects the nostrum which makes most appeal. He is utterly unaware that this shortness of breath may arise from deranged heart action and that here many possibilities exist. The pathological condition may be in the heart muscle, in the conducting mechanisms, in the site of origin of the beat or in the nervous and endocrine regulation of the organ. The arteries may be at fault, or the kidneys, or he may be right in thinking of the lungs, but unaware whether the lung tissue or the lung membrane is involved, and how the inflammatory condition, if such exists, came into being. He does not understand that a "tonic" may do harm rather than good quite apart from the neglect of the causal factor. The self-doctoring sufferer from headaches gives no thought to the possible origin of the pain in eye strain, something amiss in the nasal passages, any focus of irritation in the trigeminal area, brain tumour, circulatory or alimentary aberrancy or pyrexia; he simply swallows acetylsalicylic acid or a derivative of it and is pleased at any relief which ensues, and so the true cause of the pain goes uncorrected. Here it may be mentioned that the health regulations of the six Australian States strictly prohibit the use of salicylic acid as a preservative in food, but this is no protection to the citizen who can buy at his grocer's unlimited quantities of a powder which liberates salicylic acid when assimilated and produces in the blood a concentration of this dangerous chemical which could never be found as the result of eating sausage or other food heavily dosed with preservative.

Another form of autodiagnosis is that in which the alleged experiences of a self-cured sufferer are published in magazine article or book and beget a number of imitators. If a book is given to the public with the title "How I Got Rid of My Gall-Stones", many of the lay readers of the volume will be deeply impressed and will start on the course of treatment described, no matter if their gall-bladders contain concretions or not. It is a daily experience of the physician and surgeon, of the general practitioner and specialist, that he is called upon to deal with a condition which should have been diagnosed long before, but diagnosis and appropriate treatment have been missed because the patient was impressed with the written descriptions of an advertisement or the verbal assurances of a quack. The economic waste of patent preparations is bad enough, but immeasurably worse is the neglect of proper treatment arising from autodiagnosis. Health laws do afford some protection to the citizen and prevent him from being poisoned with mercury or arsenic or from becoming unwittingly a morphine addict, but they cannot stop him giving a name to his complaint and accepting the advice proffered by a commercial advertisement or the gossip of a neighbour. Equally mischievous is charlatan consultation, for here the diagnosis is just as inexpert and as likely to be wrong. If books on home doctoring are to be put before the public they should be confined to simple emergency first aid and should always in each instance emphasize the necessity of calling in expert help at the earliest opportunity.

Current Comment.

THE HAZARDS OF ROPE MAKING.

ROPE MAKING is one of the world's ancient trades and fundamentally its methods have changed little. In detail, of course, modern mechanical and chemical developments have had a profound effect, especially in the later stages of the process. The trade brings its own particular health hazards to those who follow it. In an informative article James A. Smiley,[1] of Belfast Ropeworks, Limited, has described the processes involved from the growing of raw materials to the packing of the various types of finished product. He points out that a modern rope works produces not only ropes with circumferences varying from half an inch to 28 inches, but also threads, twines and cords of an infinite variety. The rope works at which he is employed has manufactured over half a million different types of products including ropes for mountaineering, cords for reins, lines for anglers, ropes for gallows, cords for the drums of regimental bands, log-lines, ropes for whale-harpooning, plaited cords for window blinds, binder-twine for harvesters, and a host of other products. In Great Britain over 1500 tons of raw materials, mostly hemp, are used each week by rope works. The materials come from all over the world and, besides hemp, include jute, flax and "Nylon". Bales of hemp fibres when received are composed of bundles or "heads" which have to be separated by hand before the mechanical process begins. The separation stirs up much dust from the dried mud originally deposited on the fibres in the harvesting process. Despite considerable efforts it has not yet been possible to overcome this factor in the harvesting process, and the dust is a serious hazard to the workers in the preparing room. After the hemp has been unpacked and loosened, it is fed to machines which comb and straighten the fibre, cleaning it and forming it into continuous ribbons or "slivers". The fibre is then drawn and finally twisted into yarn on spinning machines, which also wind the yarn on bobbins ready for the making-up departments. The effect of the dust in the preparing room is the development of

[1] *British Journal of Industrial Medicine*, October, 1951.

"pousse", as it is known to the workers, a condition indistinguishable from chronic bronchitis with emphysema. The youngster commencing work has an initial period of coughing and then develops tolerance, the cough ceasing; further symptoms seldom appear for perhaps thirty years, but by that time it used to be expected that one in ten of all workers (mostly women) would develop "pousse". Smiley states that "pousse" clinically seems to resemble byssinosis as described by workers in other fields. A degree of success has been achieved recently in the control of the dust, but the hazard seems unlikely to disappear until certain essential pieces of machinery are redesigned.

In the later stages of the work problems of other kinds arise. Skin lesions occur from preservatives with which ropes and twines are treated to protect them from water, weather and (for agricultural use) insects. Tar-oil is apparently an important offender, but severe outbreaks may occur in its absence; calcium oxalate is suspected but not proved to be responsible. Oil folliculitis due to the use of mineral oils is less common than it once was; the provision of facilities for good personal hygiene, the supply of barrier creams and the transfer of the worker to another job are effective control measures. In the plaiting room the extremely high-pitched and continuous noise of the machines invariably causes nerve deafness. An attempt is being made to design a noiseless machine, but meantime new workers in this room (fortunately few are required) are recruited only from those who are already deaf. Various types of minor injury, particularly to the hands, occur in association with spinning and other processes, but a lesion of particular interest is the tenosynovitis, or *peritendinitis crepitans*, which is common amongst the braiders, who make nets. Much of this work is still done by hand and is performed with great speed and dexterity; the hazard to the extensor tendons, especially the wrist, results from the series of complicated actions of flexion, extension, pronation and supination at the wrist joint together with flexion and extension of the fingers. Early diagnosis and effective means of treatment have lessened the problem, but it will remain while machines cannot do the work of the braider. Smiley states that because of its history, the rope-making industry has been in the past very conservative, but that active steps to control the various hazards are now being taken. One hazard that he mentions as occurring from time to time will no doubt be difficult to eliminate with certainty—namely, the appearance of a hibernating snake when bales of hemp are opened in the preparing room. No injuries from this cause have occurred, but it could not be expected to increase the working efficiency of a citizen of Belfast.

THE REMOTE EFFECTS OF RHEUMATIC FEVER.

THE remote results in cases of rheumatic fever are perhaps of even greater practical interest and importance than the immediate results. They are seldom easy to predict, but it is certain that an habitually gloomy outlook is not justified. With present-day methods of treatment and particularly with recent developments in prophylaxis, the prospect is growing much brighter, but even without these the story has very often ended happily. This is well brought out in two follow-up studies, one from Denmark, the other from the United States of America. The Danish series, which has been studied by E. Arnsø, K. Brøchner-Mortensen and B. Hastrup,[1] consisted originally of 194 patients (73 males, 121 females) who were admitted to the University Hospital for Infectious Diseases in Copenhagen during the period from 1926 to 1940. All the patients included had in the original attacks typical clinical pictures of rheumatic fever with articular swelling. They were admitted to hospital during the first recognized attack of rheumatic fever in 75% of cases and during a relapse in 25%. The attack was associated with acute tonsillitis in 48% of cases and with some other infectious disease in 10%. Sixteen of the patients died in hospital. The follow-up study was made in 1949, the period of observation then ranging from nine to twenty-three years. Of the 178 patients originally discharged from hospital, 150 were still alive, 23 had died and five could not be traced. Information was available either directly or indirectly about 163 of these patients; 15 (including three who had died) had chronic rheumatic disease of the heart, and 10 (including six who had died) probably had it. Altogether 91 patients were persuaded to appear for follow-up examination; 46 had subjective cardiac symptoms, but only about half of these had more or less sure signs of heart disease of any kind. Eleven of the 91 had definite evidence of rheumatic heart disease and three had probable evidence; three of these had no complaints and all the rest were fit for work. Forty-four of the 91 patients complained of articular symptoms; 25 had objective changes, four restriction of movement only, seven periarticular changes, eight arthroses and six arthritis.

With these figures in mind we may refer to the American study, by E. F. Bland and T. D. Jones,[1] which is a follow-up over twenty years of 1000 patients who had rheumatic fever or chorea in childhood. Bland and Jones have already published a number of studies based on this group of patients, including a ten-year progress report in 1942. The present paper they describe as a second interim report; in it they summarize the major events of the two decades. It appears that on recovery from their original illness, 653 of the 1000 patients had signs of rheumatic heart disease; by the end of twenty years the signs of heart disease had disappeared in 108 cases (16%). The other 347 patients recovered from their initial illness without detectable heart disease; by the end of twenty years signs of valvular disease had appeared in 154 cases (44%). Bland and Jones suspect that when signs disappear minimal scarring persists, and there is evidence to support this, including post-mortem findings after an accidental death. The mechanism whereby deformed valves appear late is not understood. It is suggested that minimally scarred valves may form a locus for deposition of platelet thrombi, which become incorporated into the valve substance and ultimately cause deformity and stenosis. This needs further investigation. The death rate was heavy; 202 died in the first ten years, 99 in the second, a total of 301 in twenty years. Rheumatic fever and congestive heart failure caused 80% of the fatalities, and bacterial endocarditis a further 10%. Certain features present in the early stages of the disease had a grave prognostic import: among patients with a greatly enlarged heart and with congestive failure the twenty-year mortality rates were 81% and 80% respectively; with pericarditis the rate was 63%, with subcutaneous nodules 43%, with acute arthritis 27%, and, by contrast, with chorea only 12%. Recurrences of rheumatic fever or chorea took place in approximately one of five cases during the first five years, in one of ten during the next five years, in one of twenty during the third five years, and much less frequently after that. A pure form of mitral stenosis developed in 117 cases, but in only 12 has evidence of serious pulmonary hypertension appeared. Certain points about the 699 survivors add a more encouraging note to this somewhat gloomy recital. Three out of four amongst these survivors have little or no limitation; 76 served in the armed forces in World War II, and 421 children have been born to the female patients of the original group, the complications of pregnancy having been minimal. There appear to be grounds for a more hopeful attitude for surviving adults, and evidence is accumulating that the frequency and severity of rheumatic fever attacks are lessening.

It must be conceded that the American results and the light they throw on prognosis are less cheerful than what we find in the Danish paper, and apparently there are other comparable Danish series. The reason for the difference is unexplained. In either case, despite relatively encouraging features, the devastating nature of rheumatic fever is brought to the fore. We may well hope, with Bland and Jones, that in the foreseeable future new and already promising measures for the prevention and arrest of rheumatic fever may render the data reported obsolete.

[1] *Acta medica Scandinavica*, Volume CXLI, Fasciculus II, 1951.

[1] *Circulation*, December, 1951.

Abstracts from Medical Literature.

PHYSIOLOGY.

The Effects of Lung Inflation and Epinephrine on Pulmonary Vascular Resistance.

W. S. EDWARDS (*The American Journal of Physiology*, December, 1951) reports that changes in pulmonary resistance were studied in a lobe of lung retaining its natural blood supply by simultaneous optical registration of pulmonary arterial pressures and rates of venous outflow from the lobe. To avoid capacity changes on pressure flow relations, all observations were made while the lobe was maintained in states of static inflation or deflation. Both positive and negative pressure inflation of the lung was investigated. The results reported show alterations in pulmonary resistance opposite in direction to that of the pressure changes. Since these passive changes are much greater than those found to occur in peripheral systemic circuits so far studied, it was considered necessary to compare pressure flow relations over considerable ranges before and after another physiological variable was introduced. Comparison of curves representing such pressure-flow relations during pulmonary deflation and inflation has shown that pulmonary resistance is higher during lung expansion at equivalent conditions of pressure and flow. This was true regardless of whether the lungs were inflated by negative pressure in a closed chest or were expanded by positive intratracheal pressure in an opened chest. Injection of epinephrine directly into an artery of a lobe as well as into the general venous circuit produced a small but definite increase in resistance within a lung lobe at equivalent conditions of pressure and flow under static positive pressure inflation.

The Isolated Kidney and its Response to Diuretic Agents.

S. KUPFER, D. D. THOMPSON AND R. F. PITTS (*The American Journal of Physiology*, December, 1951) describe a method for the perfusion of the isolated dog kidney. When compared with values obtained *in situ*, glomerular filtration rate, clearance of para-aminohippurate, extraction of para-aminohippurate, U/P ratio of creatinine, and rate of sodium excretion are reduced when the kidney is transferred to the pump. The rate of potassium excretion is relatively unchanged, whereas the total renal vascular resistance is increased. In addition to perfusion of the blood through the lungs, the use of heparin instead of defibrination in preparing the shed blood is probably of significance in reducing the accumulation of vasoconstrictor substances. Further efforts to minimize the chemical and physical changes that occur in blood maintained *in vitro* would probably improve the functional capacities of the isolated kidney. It has been shown that both aminophylline and "Salyrgan" have direct effects upon the isolated kidney. Aminophylline dramatically reduced the total renal vascular resistance in all experiments. The changes in glomerular filtration rate were not consistent. Diuresis resulted without significant changes in glomerular filtration rate in most instances, and therefore must be attributed to diminished tubular reabsorption of sodium and water. In one instance a slight increase in glomerular filtration may have contributed to the diuresis. In several experiments no diuresis resulted. In contrast, "Salyrgan" administered intraarterially in a dose of eight milligrammes of mercury induced complete or partial renal vascular shutdown and oliguria. However, when the drug was mixed with the blood in the venous reservoir, the vasoconstrictor action was minimized. The resulting diuresis was due to diminished tubular reabsorption of sodium and water. The addition of aminophylline to "Salyrgan" solution abolished the vasospastic action noted with the intraarterial administration of "Salyrgan" alone.

Acclimatization to Cold in Rats.

E. A. SELLERS, S. REICHMAN, N. THOMAS AND S. S. YOU (*The American Journal of Physiology*, December, 1951) report that exposure of clipped rats to a cold environment (1·5° C.) results in an increased rate of oxygen consumption in less than three hours to a level about 30% above that of unclipped animals exposed to cold. This level is not maintained in unacclimatized rats, which die of hypothermia within twenty-four hours after exposure. However, in animals which have been previously acclimatized a greatly elevated metabolic rate is continued for long periods. Indirect evidence is presented indicating that the increased level of oxygen consumption of clipped over unclipped animals is due, in large part, to a greater muscular activity. Factors bearing on this are discussed.

Maintenance of Life in Bilaterally Nephrectomized Dogs and its Relation to Malignant Hypertension.

J. R. LEONARDS AND C. R. HEISLER (*The American Journal of Physiology*, November, 1951) state that most of the earlier work on animals led to the conclusion that bilateral nephrectomy resulted in no significant elevation of blood pressure. These results have been justly criticized by Grollman on the basis that the animals survived for only a short time, and, terminally, at least, were in poor condition. In a recent series of publications Grollman and co-workers presented convincing data from bilaterally nephrectomized dogs whose survival times were extended by the administration of an electrolyte-free diet and by treatment with peritoneal lavage or the artificial kidney. These studies led them to conclude that "nephrectomy results in hypertension and that the presence of intact renal tissue is essential for the maintenance of the normotensive state". These important observations were obviously in need of confirmation by other laboratories, and led to the work presented in this report. They reported that ten bilaterally nephrectomized dogs given an electrolyte-free diet of sucrose and oleomargarine supplemented by 10% glucose solution intravenously survived for an average of 6·6 days. None of these animals developed any rise in blood pressure. Fifteen similar animals treated in addition with the artificial kidney survived for an average of 9·5 days (range five to twenty days). Only one of these animals developed a significant elevation of blood pressure, and then only for the last few days of its life. Almost all the animals remained in relatively good physical condition until twenty-four to forty-eight hours before death. It is concluded that the life of some nephrectomized dogs can be prolonged by treatment with the artificial kidney, and, in agreement with the conclusion of previous investigators, that bilateral nephrectomy alone is not a sufficient condition for the development of hypertension.

Arterial, Atrial and Venous Pressure Changes in the Presence of an Arterio-Venous Fistula.

J. L. NICKERSON, F. W. COOPER, JUNIOR, R. ROBERTSON AND J. V. WARREN (*The American Journal of Physiology*, November, 1951) report that observations on the pressures in the right atrium, venous bed and femoral artery of dogs with femoral arterio-venous fistulæ demonstrate that upon opening of the fistula the arterial pulse pressure rises minimally, if at all; the pressure in the artery proximal to the fistula falls and the venous pressure rises considerably at the level of the fistula. The rise in venous pressure becomes less as the measurements are made further up the venous bed towards the atrium. Evidence for a greater emptying of the heart when the ejection is against a decreased arterial pressure suggests the sequence of events which provide the increase of cardiac output upon opening of the arterio-venous fistula.

Muscle Electrolytes and Glycogen Following Ischæmia Produced by Intraarterial Injection of Glass Microspheres.

F. A. FUHRMAN, J. L. WATSON AND J. M. CRISMON (*The American Journal of Physiology*, November, 1951) state that experimental interruption of the major blood supply to a limb by ligation of large arteries and veins does not cause significant alteration of tissue electrolyte distribution. However, severe ischæmic injury produced by tourniquet application, burning or frostbite leads to considerable alteration in the pattern of electrolyte and water distribution. Of particular interest is the accumulation in injured tissue of sodium in excess of that accounted for in the extracellular fluid. The ischæmic injury in man which most often proceeds to gangrene is associated with multiple obstruction of small arteries and arterioles rather than total obliteration of major vessels. In the experiments presented here an effort has been made to produce comparable ischæmia in animals by intraarterial injection of particles of such size that only small vessels would be obstructed. A suitable material for this purpose was found to be glass spheres. These are chemically inert, smooth and uniform in shape, and can be separated into fractions such that 50% have a diameter within 2·5μ of the mode of the distribution, and the maximum size range is only about 30μ. The authors reported that occlusion produced in this way resulted in pronounced functional impairment of the legs, delay in skin fluorescence after injection of sodium fluorescein, and gangrene of the toes. The glycogen

content of the occluded muscle was reduced two hours after injury and remained lower than that of the contralateral leg for as long as twenty-two days. Sodium and chloride concentrations in this injured muscle were increased and potassium was lost from the muscle. Sodium space in most cases exceeded the chloride space; varying amounts of sodium penetrated muscle cells after injury. In the injured muscle extracellular space is increased at the expense of intracellular space. However, calculation of the concentration of intracellular water per kilogram intracellular phase indicates that this may remain relatively constant in the injured muscle if a solid content of about 10% is assigned to the extracellular phase.

BIOCHEMISTRY.

Pantothenic Acid.

W. GOVIER AND A. GIBBONS (*Archives of Biochemistry*, July, 1951) have shown that parenteral administration of coenzyme *A* to dogs by the intravenous (femoral vein) route results in the accumulation in plasma and renal excretion of split products of coenzyme *A* active for the Kaplan-Lipmann assay enzymes. Coenzyme *A* is apparently not absorbed from the gastro-intestinal tract as such or as degradation products. Intravenous administration of coenzyme *A* to dogs resulted in the majority of cases in accumulation of coenzyme *A* in adrenals, liver and muscle tissues.

Thiopentone.

R. M. C. DAWSON (*The Biochemical Journal*, July, 1951) has shown that the free glutamic acid content of rat brain estimated after decapitation is reduced in animals anæsthetized with thiopentone. The glutamine content of the brain is slightly increased in anæsthetized rats, but this accounts for only a part of the fall in the glutamic acid level. Maintenance of the body temperature of the rats during narcosis decreases the amount by which the glutamic acid level in the brain is reduced. The glutamine and glutamic acid levels in the rat brain are not significantly changed from the normal values by alteration of the environmental temperature or by electrical stimulation of the brain. The inhibition of the respiration of minced rat brain tissue by concentrations of thiopentone has been measured. Thiopentone anæsthesia reduces the level of free ammonia found in the rat brain. Adenylic deaminose present in brain extracts is slightly poisoned by 0·04% thiopentone, while glutaminase is unaffected.

Plasma Protein.

I. A. ABDOU AND H. TARVER (*The Journal of Biological Chemistry*, June, 1951) have studied the results when plasma labelled by feeding serine-β-C^{14} from a donor rat is administered to recipient animals. After intravenous injection, the level of the label in the plasma rapidly decreases, so that it reaches half its initial level in eight hours. After three days the rate of decline becomes stabilized to one with a half-time of about three days. After intraperitoneal injection, the level of the label in the plasma reaches its maximum after about six hours, and thereafter behaves as the intravenously injected material. Oral administration of plasma does not result in strong labelling of the plasma in the recipients. After intravenous administration, the rate of labelled carbon dioxide production reaches its maximum in about two to three hours. After intraperitoneal administration, the rate of labelled carbon dioxide production reaches its maximum after three to four hours and thereafter behaves in the same manner as after intravenous injection. After oral administration the rate of labelled carbon dioxide production reaches its maximum very early in the experiment and thereafter shows a rapid rate of decline, which brings it below the intravenous rate in twelve hours. Little of the label is lost into urine or fæces after plasma is given by any one of the three routes. The label appears immediately in all the tissues. Tissues active in protein breakdown, rich in cathepsins, and active with respect to protein turnover accumulate more of the label than does muscle. The data give support to the concept that plasma protein is broken down rapidly in a few tissues.

Diabetes.

D. STETTEN *et alii* (*The Journal of Biological Chemistry*, October, 1951) have studied glucose production and oxidation in normal and diabetic rats. By means of continuous intravenous injection of a solution of C^{14}-glucose into anæsthetized rats, constant levels of specific activity of urinary, and presumably circulating, glucose have been attained in untreated, phlorhizinized, and alloxan-diabetic rats. From the specific activities of injected and excreted glucose and the rate of injection, the rate of formation of glucose from sources not derived from infused glucose has been calculated. This rate, found to be 14 or 15 milligrammes per 100 grammes of rat per hour in the normal and in the phlorhizinized animals, exhibited a statistically barely significant increase to about 21 milligrammes per 100 grains of rat per hour in the alloxan-diabetic rats. From the specific activity of the exhaled carbon dioxide a pronounced decrease in the rate of total oxidation of glucose was observed incident to phlorhizin or alloxan administration. The normal rat was estimated to oxidize 11·5 milligrammes of glucose per 100 grammes of rat per hour, whereas the phlorhizinized and alloxan-diabetic rats oxidized 2·6 and 4·5 milligrammes per 100 grammes per hour respectively.

Ketosis.

C. H. BEATTY AND E. S. WEST (*The Journal of Biological Chemistry*, June, 1951) have reported that the administration of various precursors of oxalacetic acid (succinic, malic, α-ketoglutaric, aspartic, glutamic and *cis*-aconitic acids and alanine) and oxalacetic acid itself caused a decrease in total urinary ketone body excretion in rats made ketotic with butyric acid. Inositol also caused a decrease in ketonuria in animals fed butyrate. The tricarboxylic acid cycle inhibitors, trans-aconitic and malonic acids, caused a decrease in ketonuria, while sodium fluoroacetate, also a tricarboxylic acid cycle inhibitor, increased ketone body urinary excretion in rats fed butyric acid. The results reported are regarded as additional evidence supporting the operation of the tricarboxylic acid cycle in the intact animal, and as evidence *in vivo* for the theory of fatty acid oxidation via the tricarboxylic acid cycle. The data support the theory that ketosis is related to a lack of oxalacetic acid.

Cortisone.

L. LAYTON (*Archives of Biochemistry*, June, 1951) has reported that high dosages of cortisone inhibited the synthesis of chondroitin sulphate in the tissues of the rat. He states that this may be secondary to the alteration in protein metabolism indicated by a large negative sulphur balance. A failure to form hexosamines may be partly responsible for the disappearance of chondroitin sulphate from tissues; a simultaneous decrease in all the mucopolysaccharides would be expected.

Antibiotics.

L. E. CARPENTER (*Archives of Biochemistry*, June, 1951) has shown that aureomycin, terramycin, penicillin, streptomycin and chloramphenicol stimulate the growth of pigs at the level tested under the environmental conditions imposed. All the antibiotics except chloramphenicol controlled diarrhœa. Vitamin B_{12} and streptomycin given alone, either orally or parenterally, stimulated the growth of weaned pigs. No greater effects were obtained when both were given together. The vitamin had no effect on incidence of diarrhœa, but streptomycin administered either orally or parenterally controlled diarrhœa.

Cervical Mucus.

L. B. SHETTLES *et alii* (*The Journal of Biological Chemistry*, October, 1951) have reported that 75% to 80% of the polysaccharides in human cervical mucus from the mid-cycle consists of a neutral mucopolysaccharide which contains as constituents methylpentose, galactose and hexosamine. The ratio of galactose to hexosamine is 1:1, and the content of methylpentose varies between 15% and 20% of the total mucopolysaccharide. The composition of the mucopolysaccharide is very similar to that of human blood group substances.

Insulin.

F. BINKLEY *et alii* (*The Journal of Biological Chemistry*, September, 1951) have found that the levels of glutathione of liver tissue of rats were significantly reduced after the administration of insulin. Levels of glutathione in the blood of rabbits were increased after the administration of insulin. The administration of glutathione to rabbits was followed by a decrease of levels of glutathione of the blood.

Alloxan.

D. SELIGSON AND H. SELIGSON (*The Journal of Biological Chemistry*, June, 1951) have examined the nature of the decomposition of alloxan in plasma and buffered solutions. They state that alloxan is converted to alloxanic acid under the conditions employed, and this reaction is complete or nearly so, and more rapid with increasing alkalinity. If alloxan plays any part in *diabetes mellitus*, its role might be further elucidated by the study of its more stable conversion product, alloxanic acid.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XVIII.

DIAGNOSIS OF PAIN IN THE CHEST.

COMPLAINTS of pain in the chest are among the commonest symptoms for which the clinician is consulted. In most cases the underlying cause is discovered without much difficulty, but by no means is this invariably so.

Of the many diseases which produce pain in the chest, the two which are by far the most frequently met with are the left breast pain associated with psychoneurotic ill health, and disease of the coronary arteries. It is of the utmost importance to distinguish between these two, because a mistake in diagnosis may lead to chronic, and even life-long, invalidism, and quite unnecessary suffering. Unfortunately, this discreditable state of affairs is encountered only too often. It is proposed to discuss these two conditions first, and then to review some of the other causes of chest pain more briefly.

Left Breast Pain.

Although the psychosomatic disturbance of left breast pain is widely known, it is still sometimes mistaken for organic heart disease.

The syndrome was formerly referred to as "pseudo-angina". In 1937 Bourne suggested *"angina innocens"*. In America it is known as neuro-circulatory asthenia, but this corresponds more to Lewis's "effort syndrome", a more fully developed stage. The terms "autonomic imbalance" and "phrenocardia" are now rarely used.

Middle-aged women and young adults of both sexes are the main sufferers, but no age group is exempt.

Chest pain, breathlessness and palpitation are the chief symptoms, suggesting heart disease, both to patient and doctor. Nervousness, insomnia and fatigue are also mentioned in the history. For the correct assessment of pain, breathlessness and palpitation, sufficient time must be given to careful interrogation and examination of the patient. The spectre of iatrogenic heart disease must be kept in mind, but not to the exclusion of the possibility of coronary disease.

The pain is quite different from that of coronary insufficiency. It is a dull ache, lasting for hours or days, and interspersed with sudden, sharp stabs, of brief duration.

The site of the pain is in the region of the left breast or just below, and may occasionally radiate to the left shoulder and left arm. It is precipitated by emotional upsets and fatigue, rather than by exertion, although the latter may aggravate the pain.

Exertional dyspnœa may be evident, but what the patient usually means by "breathlessness" is a subjective sensation —"I cannot get a good breath", or "I have to keep trying to take deep breaths", or "I have to keep sighing"—this sensation occurs without exertion. The term "palpitation" is used to describe tachycardia, consciousness of the heart beats, or premature beats ("fluttering in the breast").

Although the diagnosis is usually clear from the history alone, examination is essential, not only to exclude organic disease, but to reassure the patient that the clinician is certain of his ground. These people are very "suggestible", and have come for consultation with preconceived conviction that they have heart disease.

The examination will probably reveal objective signs of anxiety—an apprehensive manner, tremor, clammy hands, and tachycardia. The heart will usually be normal, but often a soft systolic murmur will be heard. This may be late systolic in time, and may change in intensity, or disappear, when the heart rate settles down, or when the patient changes his posture. An occasional premature beat and some increase in the systolic pressure are common, and finally, tenderness in the superficial structures over the site of the pain may be present.

After the examination, the patient is firmly told that his symptoms are not due to heart disease. He should then be encouraged to discuss his worries and conflicts, and when the cause of his anxiety is clear, the physician is in a position to offer a simple explanation of how emotional upsets produce the symptoms that have been related.

Beware of telling the patient that there is nothing the matter. It is untrue, and will do harm. Many doctors suffer from this syndrome, and require the same treatment.

If, however, the examiner is uncertain of the diagnosis, he should press for a full cardiological examination. This should not be postponed, otherwise the patient will be more convinced than ever that he has some serious disease. This uncertainty will be infrequent, and due to the difficulty of obtaining a clear description of the pain, coupled with knowledge that the pain of coronary insufficiency is, in about 5% of cases, located in the left side of the chest.

There is one variant of the left breast syndrome which, although uncommon, is worthy of mention, and which is initiated by trauma. It occurs when a man finds it necessary to make a sudden violent muscular effort, perhaps to save himself from being hurt. He feels a sudden acute pain in the left side of his chest. He becomes faint, and fears he has strained his heart. Fear causes tachycardia, and if the physician who examines him shows any doubt about the diagnosis, a vicious circle is set in motion, with the development of effort syndrome.

The cause of the pain is rupture of some of the fibres of the pectoral muscles at their attachment to the ribs. When the damage occurs on the right side, the fear of "heart strain" does not arise.

Finally, it must be remembered that the syndrome of the left breast pain not infrequently complicates organic heart disease of any type.

Disease of the Coronary Arteries.

Angina Pectoris.

The classical substernal pain of *angina pectoris*, described as crushing, squeezing or like a heavy weight, is unmistakable. The radiation of the pain across the chest, perhaps up to the neck and jaws, and down the left, or both arms, and the fact that it is brought on by exertion, particularly walking, and relieved by standing still, make it unlike any other condition.

However, quite a number of people cannot describe their sensation clearly, or give a confused account, often distorted by their own interpretation of the cause of the pain. In others, the pain itself is atypical, and located in the jaws, elbows or wrists, with little or no reference to the chest. The relation of the pain to effort gives the clue.

In contrast to patients suffering from left breast pain, these people are more likely to attribute their symptoms to indigestion than to heart disease, because of their observation that it often comes on soon after eating. This is so, but the angina is produced by walking back to work with a full stomach, coronary insufficiency being brought about with less effort under these circumstances when blood is pooled in the splanchnic area.

Too much significance should not be attached to the statement that the degree of pain is proportional to the physical effort, because sufferers from angina often deny that it is so, maintaining that on some days they can do far more than on others.

Variable factors are responsible for this, coronary insufficiency occurring more readily, for example, on cold or windy days, during exhaustion, during a mild infection, or after eating.

It must be emphasized that examination of a patient who gives a typical history of angina of effort will give entirely normal results in about one case in five. An X-ray examination of the chest and an electrocardiogram may also show no abnormalities.

Hypertension will be found in about half the cases, and cardiac enlargement will occur only if aortic disease, hypertension, or an old infarct is present. Thickening and tortuosity of the brachial arteries may be found, but are of no help in diagnosis.

Although atheroma of the coronary arteries is the cause of the trouble in 90% of all cases, aortic valvular disease, both stenosis and incompetence, is important, and the lesion may be syphilitic, rheumatic or calcareous. The infrequent, but curable, causes of angina should not be forgotten—toxic goitre and a severe grade of anæmia. The great importance of the history in this disease is obvious.

Coronary Occlusion.

The three main features of coronary occlusion, substernal pain, shock and cardiac failure, are sometimes all present together, but usually one of the three predominates.

A patient who has previously suffered from angina of effort knows that the pain of occlusion is different. It is more intense, lasts longer, and is not relieved by trinitrin. He feels ill, and may vomit and sweat.

Pain is most often the presenting symptom, but occasionally this passes off, and if the first examination takes place later in the attack, shock may predominate. In a small group—10% or less—there is a complaint not of pain, but of

XUM

sudden and acute breathlessness, due to cardiac failure and pulmonary congestion. The leads to the diagnosis in this group are the suddenness of the dyspnœa, the absence of any previous heart disease, and the normal exercise tolerance up to the time of the attack.

The majority of attacks of coronary occlusion come on suddenly, during rest, but it is not uncommon during exercise, particularly in those previously suffering from angina. Prodromal symptoms are sometimes present; these are described as pains of anginal type, but lasting longer, perhaps up to an hour, and recurring for one, up to fourteen, days prior to the major attack.

The severity of attacks varies a great deal, and what may be described as minor attacks are probably as frequent as major ones. These minor attacks are associated with little or no shock, or heart failure; the pain is less intense and of brief duration, and the patient continues his daily routine, and does not report to his doctor, and may never do so. If the pain recurs, or he becomes breathless, and an electrocardiogram is taken, evidence of a previous occlusion is found. Therefore, if a patient says that he has had some substernal pain, but is now free, even if he looks well and active, it is wise to have an electrocardiogram taken. The test will give positive results in practically all cases of occlusion, provided a sufficient number of precordial leads are used.

In the minor attacks, the usual reactionary fever, the leucocytosis, the fall in blood pressure, and the various forms of arrhythmia, which are so common in major attacks, may all be absent. In addition to these minor attacks, coronary occlusion leads to difficulty in diagnosis by presenting in two other ways, though more rarely: firstly, when the pain is referred to the epigastrium, and secondly, when the shock is such as to precipitate coma.

Epigastric pain, vomiting and shock, particularly when contraction of the abdominal muscles is evident, suggest an acute abdominal emergency, such as a ruptured peptic ulcer, acute pancreatitis or acute cholecystitis. Signs of pulmonary congestion, dyspnœa, arrhythmia and a tender, enlarged liver, from acute cardiac failure will assist in diagnosis, but it is sometimes difficult. An electrocardiogram is invaluable.

When a patient with coronary occlusion is first seen in coma, albuminuria and glycosuria are sometimes found, suggesting the possibility of uræmia, diabetes or meningitis. If the patient does not die in coma, he usually recovers consciousness fairly quickly, and is able to remember that acute chest pain preceded his syncopal attack.

Pulmonary Embolism and Infarction (Acute Cor Pulmonale).

In severe cases of pulmonary embolism, when the embolus is such as to occlude a large pulmonary artery, the patient may die suddenly and without warning, or he may survive, his condition being one of medical emergency, resembling coronary occlusion, with pain in the chest, dyspnœa and shock.

The pain, however, is less acute, but the dyspnœa is urgent and cyanosis marked. The accompanying heart failure affects the right side, in contrast to the left in coronary occlusion. Venous distension of the jugular veins will be considerable and the second pulmonary heart sound accentuated. The electrocardiogram may show changes suggesting occlusion of the posterior type, but it is usually incomplete, and if the patient recovers, the tracing reverts to normal more quickly than it does in cases of occlusion.

In the less severe cases, involving smaller arteries, pain is often absent, or is of pleuritic type, if the infarct involves the pleura. Hæmoptysis is not common. Physical signs may be absent also, or there may be diminished air entry near the base of one lung, or friction. Careful search for evidence of thrombophlebitis in the legs should never be omitted, and information should be sought as to the possibility of any known reason for thrombosis, such as a recent operation on the pelvic organs or anus, childbirth, or a period of convalescence. Often, no such history is obtained, nor can any clinical signs of thrombophlebitis be found. Radiological examination will show an opacity in the lung fields, but this area is seldom of the typical wedge-shape described by pathologists.

Spontaneous Pneumothorax.

In spontaneous pneumothorax, if the rent in the visceral pleura is small, the lung collapses slowly, and nothing more urgent than mild dyspnœa on exertion will arise.

On the other hand, if the tear should be larger, dyspnœa will be sudden and acute, with severe pain in the chest. The pain is "tearing or rendering", and is usually felt in the side of the chest; less often it is high up in front, or beneath the scapula, and is associated with shock.

The cardinal symptoms of chest pain, dyspnœa and shock suggest coronary occlusion, but the type of pain (which is not prolonged as a rule), its situation and the absence of breath sounds on the affected side make the diagnosis clear as a rule, and it can be confirmed by X-ray examination.

Diaphragmatic Hernia.

In the middle-aged, diaphragmatic hernia may simulate coronary pain. The type and location of the pain may be identical, and it may radiate to the left shoulder. Tachycardia ("palpitation") is frequent, and occasionally dyspnœa and vomiting. The main points in favour of such pain being due to hiatus hernia are: its association with change of posture from the erect to the recumbent, the presence of dyspepsia (mainly pyrosis), the absence of any effect from exertion, and the failure of trinitrin to afford relief.

The symptoms are often somewhat vague, and of mild degree, and the pain or discomfort is lower down, in the epigastric area. A barium meal examination, with the use of a special technique, is the only means of detecting the presence of hiatus hernia.

Fibrositis of the Chest Wall.

Fibrositis of the chest wall is likely to lead to difficulty in diagnosis only when the area affected lies over the precordium. The sharp nature of the pain, its persistence for days or weeks, and the local tenderness, particularly if small tender nodules can be felt, are usually distinctive, when the lungs and heart are found to be normal. The relief obtained by infiltrating the tender area with a local anæsthetic will help to confirm the diagnosis.

Lobar Pneumonia.

The abrupt onset and acute course of pneumonia are less often seen nowadays, the low-grade type being common, especially in the aged. The symptoms are sometimes indefinite, and the chest pain is not typical of pleurisy, so that the triad of symptoms—pain in the chest, dyspnœa and tachycardia—may once again be presented.

The pain is seldom an outstanding symptom, and when physical signs in the lungs fail to appear for two or three days, the picture resembles cardiac failure. The cough sputum and "catching" type of respiration will usually give a lead, and radiological examination will show early changes before physical signs can be detected.

Other Conditions.

There are, of course, many causes of chest pain besides those which have been discussed, for example, the root pain of *tabes dorsalis* and diseases of the thoracic vertebræ, carcinoma of the lungs, aortic aneurysm, and dissecting aneurysm. It is important to make a diagnosis in such cases, but the greatest good to the greatest number will be done by saving people from supposed heart disease that does not exist.

R. WHISHAW,
Hobart.

Medico-Legal.

KING VERSUS STENING.

IN an action heard recently in the Supreme Court of New South Wales by Mr. Justice Kinsella and a jury the Plaintiff, Miss Agnes King, a nursing sister, claimed damages for negligence and breach of contract from Dr. Warwick Stening. The jury found a verdict for the Plaintiff and awarded her damages in the sum of £3760 17s. 5d.

The Plaintiff sued both in negligence and in breach of contract, but the substance of both counts was identical. Her principal particulars of negligence, including issues raised during the trial, were shortly as follows: (i) That an operation for fracture of the neck of the femur was improperly performed. (ii) That she was permitted to get out of bed at a time too soon after the operation had been performed. (iii) That the Defendant permitted the Plaintiff to be discharged from hospital on the 9th June 1948 when on the 8th June 1948 she had reported pain, heat, swelling and absence of active movement in the affected limb. (iv) That the Defendant permitted the Plaintiff to be discharged from hospital without the fitting of a caliper. (v) That the Defendant delayed unduly in prescribing a caliper for the Plaintiff, and finally prescribed a caliper unsuitable for the purpose for which it was intended. (vi) That the Defendant

XUM

indicated to the Plaintiff that she might bear some weight on her injured leg.

There were, in addition, certain other issues raised alleging that the Defendant had failed from time to time to prescribe the correct treatment for the Plaintiff, but these were less material than those set out above and were not strongly relied upon by the Plaintiff.

The Defendant's defence was a denial of the negligence alleged.

The Plaintiff herself gave evidence in the following terms: On the 27th April 1948 she had fallen at home and was admitted to Sydney Hospital on the following day, where after clinical and X-ray examination she was informed that she had sustained a fracture of the neck of the femur. After initial treatment in a fracture bed the Defendant performed an operation on the 7th May 1948, when the fracture was fixed with a Smith-Petersen nail. She was next visited by the Defendant on the 11th, and then on the 18th of May, when the Defendant ordered the sutures to be removed and ordered that she could have leg exercises under the care of a physiotherapist. On the 25th she again saw the Defendant, who said that she could get up on the following day, that is to say, eighteen days after the operation. She did not see the Defendant again until Tuesday the 8th June. On that day she told the Defendant that her leg was sore and swollen, and that she had been having a daily bath for the last four days. She suggested that the Defendant might prefer to see a further X-ray film before she left. The Defendant said that she could be X-rayed before she left and indicated that she could go by Friday. A few minutes later the sister in charge of the ward, Sister Simpson, after speaking with the Defendant, told the Plaintiff that she could go home the following day, Wednesday, and that the Defendant would see the X-ray films when he next came round. The Plaintiff was X-rayed the next morning (9th June) and discharged in the afternoon.

After getting home the Plaintiff (who lived with her brother) rested most of the time, and was unable to do much for herself. On the first day she had a bath, but found this too difficult and thereafter had showers. Pain in her limb gradually increased, and after a telephone conversation with the Defendant, an appointment was made for the 13th July at the Defendant's rooms. There the Defendant examined the leg and said that the clinical signs were good but that some pain was to be expected. An X-ray was taken after the examination and a day or two later the Defendant phoned the Plaintiff and told her that the X-ray film disclosed signs of some absorption "in that leg" and advised her to have a caliper fitted. This she did and again visited the Defendant's rooms on the 20th July when the Defendant checked the caliper. On that day the Plaintiff requested a second opinion on the X-ray film of the 13th July. The Defendant suggested that a second opinion be left for three weeks. The Plaintiff found the caliper uncomfortable and so informed the Defendant on the telephone. A further appointment was made for the 11th August. An X-ray was taken then and the Defendant said that there was no improvement in the Plaintiff's condition, and asked whom else she would like to see. (The Plaintiff alleged throughout this part of her evidence that it was always she who had suggested a second opinion and that the Defendant seemed reluctant to arrange such a consultation.) Subsequently, an appointment was made with Dr. L. Macdonald on the 30th August. When the defendant informed her of this appointment he told her that the X-ray taken on the day of her discharge, that is, 9th June, disclosed signs of early bony union.

Subsequently in October 1948 an osteotomy with the insertion of a pin and plate was performed by Dr. Hamilton at the Royal North Shore Hospital. After the operation she was splinted in a double long leg plaster spica. About the 31st May 1949 plaster was removed and she was discharged with a caliper. This caliper was some four inches longer than that fitted by the Defendant. In January 1950 she ceased to use the caliper and commenced to move about with a crutch and a stick. Her hip was still unstable and she was unable to walk without a crutch and stick, and unable to carry on her occupation as a nurse.

In cross-examination the Plaintiff said that her principal complaints against the Defendant were that he had allowed her to get up too early and to be discharged from hospital too soon. When she first got up she was told not to put her full weight on the injured leg: she alleged that she should not have been allowed to put any weight on it. She alleged also that she should not have been permitted to have a daily bath, whereas on the 8th June the Defendant told her that she could continue to do so, but should not put undue weight on the leg unnecessarily. She alleged that the Defendant had told her to put some weight on the leg but not to bear her full weight on it. She agreed that between leaving hospital (on the 9th June) and the 13th July, she would have put her full weight momentarily on the leg only when having a shower, going to the toilet, pulling up her bedcovers and getting into bed.

Dr. Patrick Howard Speight, Medical Officer to the Queensland Insurance Company, gave the only other evidence for the Plaintiff. He stated he had known the Plaintiff for many years and was her medical adviser. He had had experience of orthopædic cases but was not an orthopædic specialist. He was shown the X-ray film taken on the Plaintiff's admission to hospital and said that it disclosed a fracture, high up, of the neck of the femur, in a good position. It was a "sliding" fracture, that was to say, the type of fracture where the fragments tended to slide apart under any pressure. In such a case, it was of importance to see that no undue weight was placed on the limb to bring about a "slide". Extra care would also have to be taken to prevent a necrosis of the tissue near the base of the head, because of the poor blood supply. Too early weight bearing on the fracture site could cause a necrosis. In a fracture such as the subject one no weight should be borne for at least three months and probably not for six months.

In his view Sir Reginald Watson-Jones was probably the greatest authority in the world on fractures and in particular on the subject type of operation. The following statement from Watson-Jones's treatise on "Fractures of Bones" (1943 Edition) at page 642 was put to him:

> Many surgeons allow weight-bearing within two or three weeks of operation, but this practice is dangerous. . . . Weight bearing should not be allowed earlier than two months after operation in low fractures and not before there is radiographic evidence of union in high fractures; . . .

He had had this passage in mind and agreed with it. A statement in an article at page 190-191 in the "Year Book of Orthopædic Surgery" for 1950 was put to Dr. Speight and read as follows:

> Weight-bearing should be prohibited until there is evidence of solid bony union. . . . Post-operatively . . . an ambulatory Thomas Splint is made, as unsupported weight-bearing is avoided for six months or until trabeculation of the fracture is clearly visualised on the rays.

Dr. Speight agreed with this statement, and went on to say that in his opinion weight bearing upon a high fracture of the subject kind would have deleterious effects. It would cause a tearing of the tissues at the fracture site, a flooding of blood into the fracture site which would disturb the new bone cells, and would start an aseptic necrosis: absorption would follow. In addition weight bearing would cause upward displacement of the shaft, in turn causing the pin to be torn out of the head. Disengagement of the pin would be more likely to occur if it had originally been inserted in a low position. The X-ray films taken at the first operation on the 7th May 1948 showed that the pin had been inserted in a low position. The original fracture could not have impaired the blood supply to the head of the femur, as these X-ray films would then have shown signs of necrosis in the head, whereas the head appeared to be vital. The X-ray films taken on the 9th June 1948 (the day of the Plaintiff's discharge from Sydney Hospital) were shown to Dr. Speight. In his view they disclosed that the pin was cutting out of the head and that there was no evidence of any bony union. In the light of such X-ray films he would not have discharged the patient. In particular, since there was no sign of union, no weight bearing could be allowed. An X-ray film taken on the 13th July 1948 appeared to show that the pin was almost out of the head: vitality of the head and the upper end of the femur were still very evident. There had been some upward displacement of the shaft which was accountable to too early weight bearing, assisted by the low position of the pin. He could not be positive as to the presence of absorption either at 9th June or 13th July. Any degree of absorption present would have been caused by weight-bearing. Further the X-ray of 13th July called for some immediate steps to be taken, that was to say, an immediate osteotomy. The X-ray films taken on 11th August 1948 showed that the pin was completely disengaged, that there was almost complete absorption of the neck and upward displacement of the shaft. There was then less chance of a successful further operation. An X-ray film taken after the operation performed by Dr. Hamilton in October 1948 showed that the head was still vital. An X-ray taken on the 14th March 1952 showed that the plate inserted by Dr. Hamilton had fractured: there was further upward displacement of the shaft and no sign of bony union; the head was still vital. He did not think

that the wearing of a caliper as from the 9th June 1948 would have been of much assistance.

In cross-examination Dr. Speight agreed that if the blood supply (to the head of the femur) was sufficiently damaged at the time of the fracture, union would be most unlikely to follow. He took the prohibition against weight bearing he had expressed as meaning that no weight whatsoever should be placed on the injured leg. He was prepared to follow the practice of Watson-Jones in cases of this sort, and was not really familiar with any other authorities.

This was the Plaintiff's case. The issue as to improper performance of the operation had not been included in the particulars of negligence served before the hearing commenced and was not raised until the second day of the trial. The expert witnesses called for the Defendant all agreed that the operation had been properly performed and the pin inserted in an entirely satisfactory position. At the conclusion of the Defendant's case the Plaintiff called evidence in reply to support the allegation that the operation had been improperly performed, and this evidence will be set out later.

The Defendant then gave evidence. Prior to performing this operation he had performed the same operation on some 15 to 20 occasions and had attended or assisted at up to about one hundred similar operations by Dr. John Hoets. He described the operation which in his view was a very difficult one. He described the general course and system of treatment in Sydney Hospital, and added that when on the 25th May he told the Plaintiff that she could get up the following day, he instructed her to take no unsupported weight on the injured leg. When he saw her on the 8th June he perused the progress report and noted that on the 28th May the Plaintiff had complained of gross swelling over the site of the pin, and that a hæmatoma had been provisionally diagnosed. A clinical examination, however, failed to disclose any sign that this condition was still present, and Dr. Firkin, the Junior Resident, informed him that the pain and swelling complained of had been nothing out of the ordinary. He then directed that the Plaintiff could leave hospital the following day subject to the routine X-ray being satisfactory. The X-ray films were taken the next morning, when he saw them, and instructed Dr. Firkin that the Plaintiff could leave. On the previous day he had inquired about her home conditions which appeared to be satisfactory.

On the 15th June the Plaintiff had written him a letter thanking him for his attention, and to this letter he replied. The letter made no complaint as to her condition or of any pain. Following a telephone conversation subsequently, an appointment was made for the 13th July at his rooms. On that day a clinical examination disclosed almost full range of passive movement, no pain and no shortening of the limb. He then sent her for X-ray examination, and saw the films the following day. These showed that absorption had occurred in the neck of the femur, and that there had been some upward displacement of the shaft. Absorption was caused by deficient blood supply, which was itself due to damage occurring at the time of fracture. The pin, however, appeared still to be engaging the head of the femur. He thought that the possibility of union was remote, but that it was quite justifiable to wait and see how things progressed rather than subject the Plaintiff immediately to a further major operation. Accordingly he ordered a caliper and advised the Plaintiff to rest as much as she possibly could and to return in a month's time. Meanwhile the Plaintiff returned wearing the caliper which he checked: it appeared to fit perfectly.

On the 11th August he saw the Plaintiff again and X-ray films taken that day disclosed that further absorption in the neck of the femur had occurred and that there had been further upward displacement of the shaft. The pin had moved up in the head of the bone. He advised a further operation. The Plaintiff was unwilling to return to Sydney Hospital and accordingly he arranged for her admission to the Royal North Shore Hospital.

X-ray films taken subsequent to the second operation showed progressive degeneration of the head of the femur. In his view blood supply to the head of the bone was so interfered with at the time of her original injury that union could not occur.

On the 8th June 1948 the Plaintiff had not complained of any pain and swelling and had not told him that she had been having daily baths. To his knowledge she had never had a bath while in Sydney Hospital, save for sponging in bed. The Plaintiff had never asked him for a second opinion.

Referred to the term "weight bearing" as used by Watson-Jones, the Defendant took this to mean taking full body weight, and pointed out that up to about 1940 some orthopædic specialists considered early full weight bearing an advantage. Watson-Jones was thus writing with this practice (then generally discredited) in mind.

In cross-examination the Defendant denied that weight bearing had caused the pin to cut out. The reason was softening and absorption of the head due to inadequate blood supply. The deficiency of blood supply was caused by the original fracture. It was not normal practice to fit a caliper on discharge from hospital, in this type of case. He agreed that the X-ray film of the 9th June showed some absorption at the neck, but considered that it was quite safe to allow the Plaintiff to go out. She should not bear her full body weight on the leg, but it would do no harm to rest the foot on the ground in order to balance herself. In his view the amount of weight bearing described by the Plaintiff as having taken place between her discharge from hospital on the 9th June and the 13th July could not have caused the condition disclosed in the X-ray film taken on the latter date.

Dr. Thomas King said he had performed about 600 operations with the Smith-Petersen pin, and had been one of the pioneers in this type of operation. He had found that union was achieved in only about 70% of cases: failure was due to interference with the blood supply to the head and neck of the femur caused at the time of the fracture. He understood that by weight bearing (in the passage put to Dr. Speight) Watson-Jones meant bearing full body weight. As to post-operative treatment, in this case he would have had the Plaintiff out of bed in a fortnight, and home in a fortnight. He did not ordinarily prescribe a caliper.

In his view the X-ray films taken on admission to Sydney Hospital on the 28th April 1948 showed that the fracture was comminuted with serious interference to the blood supply. The X-ray film of 9th June 1948 (taken on discharge) showed that the pin was beginning to cut out in front; he disagreed with the radiologist's report to the effect that there were signs of early bony union, and thought that union was then already unlikely. The X-ray films of 13th July showed further degenerative changes, the onset of avascular necrosis and considerable absorption of the neck. The pin was still engaging the head but had cut out further. By the 11th August the changes were more marked. The head was no longer vital. The X-ray taken on 14th March 1952 showed that the neck had completely absorbed. The signs were those typical of an avascular necrosis.

He expressed agreement with the Defendant's treatment of the Plaintiff and thought that the delay in the performance of the osteotomy had made no difference. In his view the X-rays of the 9th June showed that thereafter nothing could have been done to effect union, certainly in the light of the techniques available in 1948.

In cross-examination he said that in his opinion what the radiologist's report of the 9th June 1948 referred to as signs of early bony union were really only splinters of bone from the fracture. There was no danger in a patient bearing some weight on the injured limb, although it was certainly deleterious to permit the bearing of full body weight at too early a stage. Full weight bearing could cause a failure of union and absorption. The position of the pin was entirely satisfactory.

Sister Simpson, who had been in charge of the ward to which the Plaintiff had been admitted, denied that the Plaintiff had ever had a bath in a bath during her stay in hospital. When the Plaintiff had got up on the 26th May she had been instructed not to place her full weight on the injured leg. The Defendant had ordered that the Plaintiff be X-rayed before discharge.

The Plaintiff, to her knowledge, had never complained on the 8th June of pain and swelling, and on the day of her discharge told her that after a couple of days she intended to drive herself up to Gosford. Sister Simpson told her that this would be a dangerous thing to do.

Mr. J. M. Smith gave evidence to the effect that he had fitted the Plaintiff with a caliper after her discharge from Sydney Hospital. As far as he remembered, the caliper had fitted properly.

Dr. C. L. Firkin had been a junior resident medical officer at Sydney Hospital in 1948, and remembered the Plaintiff. Most of the entries in the Plaintiff's progress report had been made by him. Any complaint of pain by the Plaintiff would have been written into the progress report. He noted that there was no such entry after the 28th May. The Plaintiff had never, to his knowledge, had a bath in a bath while in hospital. The Defendant had, on the 8th June, directed that the Plaintiff be X-rayed before discharge on the following day. On the 9th June the X-ray film was taken and late that morning the Defendant notified him that the X-ray was satisfactory and that the Plaintiff could be discharged.

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In cross-examination he admitted that he had made an entry dated the 5th June to this effect: "Is going home on Wednesday next." He said, however, that this was a provisional decision only and subject to the result of a final X-ray examination.

Dr. W. D. Sturrock had seen Watson-Jones operate and did not get the impression that he found this type of operation easy. He agreed that Watson-Jones had meant by weight bearing, bearing the full body weight. In his opinion, Watson-Jones had intended in the passage under discussion from his book written in 1943 to discourage the practice, then still current, of allowing patients to walk immediately after the insertion of a Smith-Petersen pin. Partial weight bearing would be unlikely to cause harm. In his view the deterioration shown in the X-ray films of the 13th July was not caused by weight bearing, having regard also to the fact that the Plaintiff denied that she had done any more after her discharge from hospital on the 9th of June other than bear partial weight only, and that intermittently. In his opinion, those X-ray films disclosed that the fact was that damage to the blood supply at the time of the injury was such that union was never possible.

Dr. L. J. Woodland had seen Watson-Jones operate and was familiar with his post-operative treatment. He (Watson-Jones) would get his patients up on crutches and would permit them to bear some weight on the injured leg. In Dr. Woodland's experience, in this type of operation, one could expect one-third good results, one-third non-union, and in the remaining third the onset of crippling arthritis within two to ten years. The principal cause of non-union was avascular necrosis and resulting absorption due to interference with blood supply. This interference with blood supply was determined at the time of the injury. He had examined the Plaintiff in November 1951 and had then expressed the view that the ultimate result had been determined at the time of injury. He had seen the X-ray film of the 9th June and that disclosed that the pin was quite satisfactory, but that there was some absorption and perhaps a suspicion of a commencing necrosis. In the light of this X-ray film, together with a satisfactory clinical examination, he would have permitted the Plaintiff to go home. He thought that the X-ray film of the 13th July showed that the head was half-dead and that there was some absorption at the neck. There was some comminution, indicating that the fracture had been severe and interference with blood supply considerable. From the Plaintiff's evidence nothing she had done between leaving hospital and the 13th July would have militated against union. In his view the Defendant was quite justified in waiting for a month or so after the 13th July to see if by chance some improvement would take place. Nothing else save an osteotomy could have been done—and that could still have been done later with an equal chance of success. The pin had been inserted in a good position.

The Plaintiff then called evidence in reply, that of Miss M. Alley, who was in charge of the Sydney Hospital X-Ray Department Office. The practice was (and still is) to record all X-ray films taken in connexion with every patient. No X-rays taken in the theatre during the Plaintiff's operation (except those showing the pin in position) appeared on the record card: and no such X-ray film could be found. (This evidence was, of course, intended to prove that no X-ray films had in fact been taken to show the position of the guide wire or wires before insertion of the pin.)

In cross-examination she admitted that she recorded only those X-ray films which were in fact sent down from the theatre. X-ray films taken in the theatre could have been left there, or could have been sent down and then lost without being recorded. She knew that usually in Smith-Petersen operations there were several photographs taken of the wires. She could not understand why there were none in this case.

In rebuttal of this evidence the Defendant was recalled and said that he had inserted a guide wire of which X-ray films had been taken. Dr. Firkin was also recalled and remembered that X-ray films of the wire had been taken. He had also made an entry in the operation sheet (which was shown to him) reading: "X-ray taken; wire in good position."

Mr. K. A. Ferguson, Q.C. (for the Defendant), in his address submitted that the operation had been properly performed. Secondly, there was evidence that the Plaintiff's post-operative treatment had been quite normal and in accordance with the practice of eminent orthopædic surgeons. Evidence had been given by experts that the course of the deterioration in the Plaintiff's condition had been inevitably determined at the time of her injury. The fracture itself had caused such interference with the blood supply to the head of the femur that union in the circumstances was impossible. Further, it had been said by the expert witnesses called on behalf of the Defendant that the actual amount of weight bearing admitted by the Plaintiff could not have caused the condition found on the 13th July. In their view, no steps could have been taken by the Defendant after the 13th July (other than the osteotomy which he did in fact advise) which could have averted progressive deterioration in the Plaintiff's condition. The fact that the osteotomy had not been advised until 11th August had had no adverse effect. There was no evidence of negligence on the part of the Defendant.

Mr. G. E. Barwick, Q.C. (for the Plaintiff), said that the defence argued that non-union was inevitable from the time of the fracture. However, the radiologist's report on the X-ray films taken on the 9th June said that the appearance suggested firm union. The Defendant, too, at that stage did not think that the position was hopeless. Yet the X-ray films of the 13th July showed marked deterioration, and as at the 11th August it was probably right to say that any chance of union was gone. The issue was plain. There had been lack of care either in the original operation or in discharging the Plaintiff from hospital and sending her home free to move about and to put weight, however cautiously, on the injured leg. It had been conceded that weight bearing could cause absorption and a failure of union. The X-ray films of the 13th July which showed upward displacement of the shaft were entirely consistent in his submission with the explanation that this displacement and consequent non-union had been caused by weight bearing. He referred also to the entry in the progress report, dated the 5th June, and contended that this showed that the decision to discharge the Plaintiff had been taken before the Defendant could be satisfied as to her condition. The Defendant had been guilty of negligence.

In his summing up His Honour first pointed out to the jury that the evidence as to the performance of the operation was all in the Defendant's favour. Subsequently on the application of the Defendant's Counsel, His Honour directed the jury that there was in fact no evidence before them of any want of care in the conduct of the operation. They would therefore ignore that issue. He went on to say that if there were two authentic schools of opinion upon one aspect of surgery and post-operative treatment, adherence to the one rather than to the other could not amount to negligence.

The principal issue before the jury was this: If the Plaintiff's condition (non-union) had been caused by damage done at the time of fracture, then the Defendant could not have been negligent. If on the other hand the Plaintiff's condition had been caused by weight bearing, then it might be that the necessity to bear weight arose only from the Plaintiff's discharge from hospital at the particular time; that was to say, if she had not been discharged she would not have had to look after herself to the degree that she had to do at home, and would not therefore have had to bear the weight she did. The jury had to consider on this point the evidence given on behalf of the Defendant to the effect that partial weight bearing would not have been harmful, and in particular, that the degree of weight bearing admitted by the Plaintiff could not have caused a failure to unite. However, there was the evidence of the radiologist's report of the 9th June which stated that there were signs of bony union commencing: yet on the 13th July any signs of union had disappeared.

If the jury was satisfied that the failure to unite was caused by weight bearing, and that this weight bearing could have been averted by a later discharge from hospital or by positive directions not to bear any weight, and if failure to take these steps showed a want of care on the part of the Defendant, then they would find a verdict for the Plaintiff.

The jury, after a lengthy retirement, returned a verdict for the Plaintiff.

Correspondence.

TREATMENT OF TUBERCULOSIS WITH ISONICOTINIC ACID.

Sir: A new antituberculosis drug has been developed in this country, and following premature and regrettable publicity in the lay Press, confusion exists in many minds as to the known facts and status of the drug.

I have read the first published reports in the *Quarterly Bulletin of Sea View Hospital* (New York) (Volume XIII, Number 1, January, 1952), and I have observed four patients treated over a period of one to five weeks in this hospital and at Johns Hopkins Hospital.

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At the outset I would emphasize the extreme caution with which this drug, a hydrazide of isonicotinic acid, is being received by those initiating the therapeutic trials. My impression is that these investigators feel that the drug is still experimental and that years will have to elapse before its clinical evaluation can be made on firm ground.

Some work has already been done with mice infected with known virulent strains of the tubercle bacillus. Some of these mice were given isonicotinic acid in varying dosage ranges, and then all the mice were autopsied to see what protection they had been afforded. The results were encouraging. For the first time a drug has been demonstrated which, at varying but non-toxic levels, affords 50% to 100% protection according to dosage against virulent bacilli. What is most encouraging is that cultures of the lungs of the protected animals have been sterile, indicating that the drug is the first *in vivo* to do permanent damage to the tubercle bacillus.

The susceptible Rhesus monkey has also been experimentally infected and treated for seventy-four days with isonicotinic acid. The autopsy revealed minute and scattered tubercles with the bacillus seen in one lymph node on section, failing, however, to grow on culture.

Clinically the drug has been administered to 92 patients at Sea View Hospital, New York, for periods up to fifteen weeks when the report was published. These patients filled the following specifications: (i) They were all far advanced, active, progressive bacillary cases with open cavitation visible on X ray. (ii) They were all pursuing an uninterrupted downhill course clinically. (iii) Other forms of therapy had failed or were not applicable. (iv) All were underweight and toxic and half were febrile.

Base-line radiological, hæmatological, urinary, renal and liver studies were done and the drug administered despite unfavourable prognosis. All other drugs were halted.

The results, as described by Robitzek *et alii* in the same issue of the *Quarterly Bulletin of Sea View Hospital*, have been dramatic. I quote: "The mortally ill patients have obtained therapeutic relief beyond anything we have ever seen with any of the chemotherapeutic agents previously utilized by us." The dosage was two milligrammes per kilogram of body weight daily in four divided doses and later increased to four milligrammes per kilogram of body weight. No serious toxic effects have been noted apart from mild symptoms ascribed to sympathetic stimulation of the autonomic nervous system. In no case was the drug suspended because of toxic reactions.

A summary of the clinical effects noted is as follows:

1. Fever abated rapidly in every instance, sometimes within forty-eight hours, and remained normal apart from transient rises in some cases ascribed to an upper respiratory infection.

2. Symptoms of toxæmia, such as weakness and anorexia, were dramatically alleviated. This is one of the earliest, most dramatic and constant effects noted.

3. Appetites increased remarkably with a marked increase in weight in all patients. This averaged two pounds per patient weekly, and there was no evidence of water retention.

4. Cough and sputum were improved in every case within two weeks.

5. Sputum studies are incomplete necessarily because of the short period of observation. However, 25% of the patients had a sputum reversion between October, 1951, and January, 1952.

6. X-ray changes are likewise incomplete. Cavity closure has occurred in some, while pulmonary destruction is so marked in most that cavity closure cannot be hoped for.

7. Extrapulmonary complications, such as laryngitis and enteritis, have all improved.

The four patients which I have observed have been three children on the pædiatric service of this hospital and at Johns Hopkins Hospital and one adult on the tuberculosis service of this hospital. They were all cases of tuberculous meningitis. In only one case has the tubercle bacillus been isolated in the cerebro-spinal fluid. The clinical course and the biochemical changes in the cerebro-spinal fluid were compatible with tuberculous meningitis. All have miliary changes in the lungs. In one case a two-year-old coloured female was treated for five months with streptomycin intrathecally and parenterally and PAS orally, when her general condition suddenly deteriorated. "Rimifon" (isonicotinic acid) was begun on February 28, 1952, and she became markedly more active, her appetite improved, and her fever fell to normal. Treatment with "Rimifon" in this case was started in an effort to save the patient's life, and apparently it has succeeded. Other cases have shown similar clinical

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 5, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	..	..	..	..	..	..	..	..
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	..	13(9)	..	..	..	2	..	15
Diphtheria	2(1)	8(7)	3(2)	1(1)	4(3)	..	..	..	18
Dysentery (Bacillary)	..	3(3)	1(1)	1(1)	..	..	..	2	7
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	..	..	..	..	11(3)	..	1	..	12
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	1	..	..	..	1
Meningococcal Infection	2(2)	5(3)	1	..	1(1)	..	..	..	9
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	7(3)	11(8)	1	14(10)	3(1)	..	..	..	36
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	2(2)	..	..	3(3)	..	..	..	5
Salmonella Infection	..	..	..	..	2(2)	..	..	..	2
Scarlet Fever	23(10)	34(20)	5(3)	3(1)	4(2)	3	..	1	73
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1(1)	..	..	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	31(25)	7(4)	29(15)	11(8)	10(7)	3	1	..	92
Typhoid Fever	..	..	1	1(1)	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	1	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

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improvement in a matter of days, which has been remarkable to those observers who were satisfied to watch the long, slow battle when streptomycin and PAS are used. I could detect no marked radiological changes in the miliary lesions in the lungs over the short period of treatment so far.

This communication is offered in case the medical profession in Australia is placed in the unenviable position of having to glean its information from the lay Press on what may be a most important therapeutic advance in the treatment of tuberculosis.

Yours, etc.,

ANTHONY J. PROUST, M.B., B.S.,
Resident, Baltimore City Hospitals.

Baltimore, Maryland,
United States of America,
March 27, 1952.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Anderson, Robert Martin, M.B., B.S., 1948 (Univ. Sydney), Box 53, Wauchope.

Middleton, Roland William Donald, M.B., B.S., 1951 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

O'Flynn, Patricia Mary, M.B., B.S., 1946 (Univ. Sydney), 106 Ryde Road, Pymble.

The undermentioned have been elected members of the South Australian Branch of the British Medical Association: Dineen, Desmond Patrick, M.B., B.S., 1951 (Univ. Adelaide); Waddy, Brian Cadwallader, M.B., B.S., 1951 (Univ. Adelaide); Magarey, Robert Richmond, M.B., B.S., 1951 (Univ. Adelaide); Daly, David James, M.B., B.S., 1951 (Univ. Adelaide); Hodge, Philip Richard, M.B., B.S., 1951 (Univ. Adelaide).

Obituary.

ERNEST ADDISON.

WE regret to announce the death of Dr. Ernest Addison, which occurred on April 19, 1952, at Devonport, Tasmania.

Notice.

THE first general meeting of the Medical Section of the National Association for the Prevention of Tuberculosis in Australia, New South Wales Division, will be held in the William H. Crago Council Chamber, British Medical Association House, Macquarie Street, Sydney, on Monday, May 12, 1952, at 5 o'clock p.m. The business will be to adopt a constitution and elect office-bearers. All members of the medical profession are invited to attend this meeting.

WE have been asked to announce that a lecture on "Milk Secretion" will be given at 7.40 p.m. on May 8, 1952, at the School of Public Health and Tropical Medicine, University of Sydney. The speaker will be Professor C. W. Turner, of the Department of Dairy Husbandry, University of Missouri, United States of America.

THE next meeting of the Section of Preventive Medicine of the Victorian Branch of the British Medical Association will be held in the Medical Society Hall, 426 Albert Street, East Melbourne, at 4.30 p.m. on Thursday, May 8, 1952. Dr. E. Forbes Mackenzie will open the discussion with a paper on "Diphtheria Prophylaxis". Representatives of the Commonwealth Serum Laboratories and Medical Officers of Health have been asked to take part in the discussion. All members of the Branch are invited to be present.

Medical Appointments.

Dr. G. H. Burnell has been appointed a member of the Council of the Institute of Medical and Veterinary Science, Adelaide.

Dr. J. G. McMahon has been appointed an official visitor to the Mental Hospital, Kew, Victoria.

Dr. N. McA. Gregg has been appointed a member of the New South Wales Medical Board, Department of Public Health.

Dr. C. E. Winston has been appointed a member of the Nurses' Registration Board of New South Wales.

Diary for the Month.

MAY 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAY 7.—Victorian Branch, B.M.A.: Branch Meeting.
MAY 7.—Western Australian Branch, B.M.A.: Council Meeting.
MAY 9.—Queensland Branch, B.M.A.: Council Meeting.
MAY 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 19.—Victorian Branch, B.M.A.: Finance Subcommittee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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